“A THREE YEAR RETROSPECTIVE ANALYSIS OF TUMOR AND TUMOR LIKE CONDITIONS OF THE OVARY IN TIRUNELVELI MEDICAL COLLEGE HOSPITAL”

DISSERTATION SUBMITTED FOR

M.D. DEGREE EXAMINATION

BRANCH III PATHOLOGY

of

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI

TIRUNELVELI MEDICAL COLLEGE HOSPITAL

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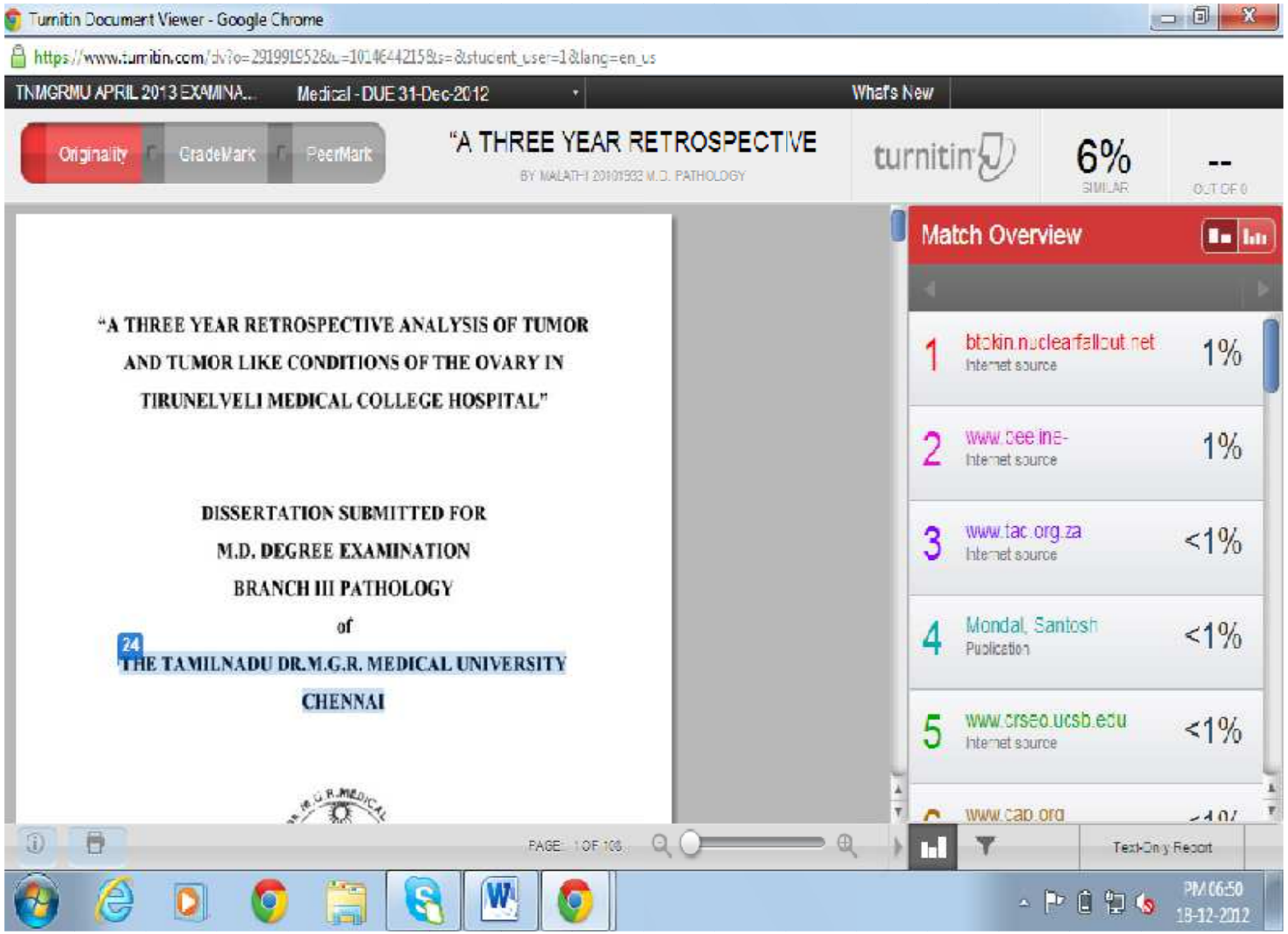
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CERTIFICATE

This is to certify that the Dissertation "**A THREE YEAR RETROSPECTIVE ANALYSIS OF TUMOR AND TUMOR LIKE CONDITIONS OF THE OVARY IN TIRUNELVELI MEDICAL COLLEGE HOSPITAL**" presented herein by **DR. MALATHI** is an original work done in the Department of Pathology, Tirunelveli Medical College Hospital, Tirunelveli for the award of Degree of M.D. (Branch III) Pathology under my guidance and supervision during the academic period of 2010 - 2013.

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CERTIFICATE

I hereby certify that this work embodied in the dissertation entitled **"A THREE YEAR RETROSPECTIVE ANALYSIS OF TUMOR AND TUMOR LIKE CONDITIONS OF THE OVARY IN TIRUNELVELI MEDICAL COLLEGE HOSPITAL"** is a record of work done by **DR. MALATHI** in the Department of Pathology, Tirunelveli Medical College, Tirunelveli, during her postgraduate degree course in the period 2010-2013. This work has not formed the basis for any previous award of any degree.

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DECLARATION

I solemnly declare that the dissertation titled **"A THREE YEAR RETROSPECTIVE ANALYSIS OF TUMOR AND TUMOR LIKE CONDITIONS OF THE OVARY IN TIRUNELVELI MEDICAL COLLEGE HOSPITAL"** is done by me at Tirunelveli Medical College Hospital, Tirunelveli.

The dissertation is submitted to The Tamilnadu Dr. M.G.R.Medical University towards the partial fulfilment of requirements for the award of M.D. Degree (Branch III) in Pathology.

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CONTENTS

LIST OF ABBREVIATIONS

WHO	-	World Health Organisation
FIGO	-	International Federation of Gynecology and Obstetrics
H & E	-	Haematoxylin and Eosin
PAS	-	Periodic Acid Schiff
CA 125	-	Cancer Antigen-125
AFP	-	Alpha Feto Protein
HCG	-	Human Chorionic Gonadotrophin
CK	-	Cytokeratin
EMA	-	Epithelial Membrane Antigen
WT-1	-	Wilms Tumour Antigen
PLAP	-	Placental Alkaline Phosphatase

INTRODUCTION

The ovaries are paired oval structures in females, developing from the gonadal ridges, and located one on each side of the uterus in the ovarian fossa. These are very complex organs in terms of embryology and histology. It undergoes cyclical changes from adolescence to menopause and gives rise to different types of cells, each of which has the capacity to produce different type of tumours[1].

Due to constant endocrine stimulation and subsequent traumatic insults by ovulation, these are the primary sites for tumour development. Different types of tumours tend to occur in different age group. Both primary and metastatic tumours occur in the ovary with variable histomorphological patterns.[2]

Ovarian cancer is one of the most common neoplasm in developed countries, ranking 7th position in incidence and 6th position in mortality[3].It constitutes about 6.6% of all malignant tumours of the female genital tract[3].In India, incidence of ovarian cancers come next to cervical and endometrial cancers among the gynaecological cancers. According to National Cancer Registry Programme of Indian Council Medical Research, the proportion of ovarian cancer varied from 1.7-8.7 in various urban and rural areas, it also showed that recent increase in incidence. Both Chennai Cancer Registry and Dindigul Ambilikkai cancer registry showed an age adjusted incidence rate of 6.0cases per 10, 0000women[4], whereas it was 16.23 in United States.[3]

Ovarian cancers area heterogeneous group of neoplasms of three main subtypes: - Surface epithelial, Germ cell and Sex cord stromal tumours with wide morphological variations. No age group is free from the tumour. In adult age group surface epithelial tumours are the commonest neoplasm constituting 65.5% of ovarian neoplasms. In younger age group, germ cell tumours are the commonest ovarian

neoplasms and constitute two third of ovarian tumours out of which, one third are malignant. Sex-cord stromal tumours can occur at any age group and are usually functional in nature. So the determination of various histological patterns is very important in diagnosis as well as prognosis of ovarian tumors[5].

Ovarian cancer has got a poor prognosis among all gynaecological cancers. The overall 5year survival rate is approximately 45% due to late stage at diagnosis[6].Unlike cervical cancer, identification of high risk population for ovarian malignancy and ideal screening method is not available.

A number of non neoplastic lesions can occur from neonatal period to postmenopausal age group. Most are functional in nature and resolve with minimal treatment. Some of the non neoplastic lesions like massive edema of ovary, stromal hyperplasia, large follicular cyst, pregnancy luteomas, and granulomatous inflammation can be confused with neoplasm clinically, intraoperatively or on morphological examination. The main aim lies in distinguishing ovarian neoplasms from the wide spectrum of non-neoplastic lesions. Despite the new techniques like imaging and genetic studies, the diagnosis of ovarian tumour is mainly dependent upon histopathological examination.

The present study is being undertaken to review in detail the different varieties of ovarian lesions in and around Tirunelveli and assess their characteristics with regards to incidence, age and histopathological appearances.

AIMS AND OBJECTIVES

1. To determine the nature of ovarian masses presented to this Department of Pathology, Tirunelveli Medical College during the last 3 years.
2. To ascertain the frequency and distribution of ovarian lesions among various age group and to correlate them with the clinical features.
3. To study the histomorphological diversity of various neoplastic and non neoplastic lesions of ovary.

REVIEW OF LITERATURE

Embryological development of the ovary:

Gonadal development occurs slowly in female embryos. Sexual differentiation of gonads is recognizable by 6th week. Ovarian differentiation is a passive event which occurs in the absence of testicular differentiation. The occurrence of familial XX gonadal dysgenesis, transmitted as an autosomal recessive trait suggests that autosomal genes are essential for human ovarian organogenesis.

Gonads appear initially as the gonadal ridges which are formed by proliferation of epithelium and condensation of mesenchyme. Primordial germ cells first appear at an early stage of development among endodermal cells in the wall of the yolk sac close to allantois, and then they reach the gonadal ridges. Before it reaches the ridges, the epithelial cells proliferate and produce irregular shaped cords called primitive sex cords. Sex cords differentiate to irregular cell like clusters. Surface epithelium of female gonads proliferates and forms second generation of gonadal cortical cords, which permeates the mesenchyme. In 4^{th} month these cords split into isolated cell clusters with each surrounding one or more primitive germ cells. Germ cells develop into oogonia and surrounding epithelial cell descendants of surface epithelium form follicular cells.[7]

Anatomy of the ovary:

Ovaries are paired structures, oval almond shaped, located one on each side of uterus close to lateral pelvic wall. They are attached to posterior aspect of broad ligament of uterus by a double fold of peritoneum (Figure 1). Each adult ovary measures 4-5 cms in length, 1.5-3.0cms in width, 0.5-1.5cms in thickness, prepubertal ovary measures about 9-17mm in length, 4-7mm in width, 2.5-5mm in depth, postmenopausal ovary measures about 2-3cm in length, 1cm in width and thickness.

The mature ovary weighs about 5-10gms whereas weight of the atrophied postmenopausal ovary is 3-6gms.[5]

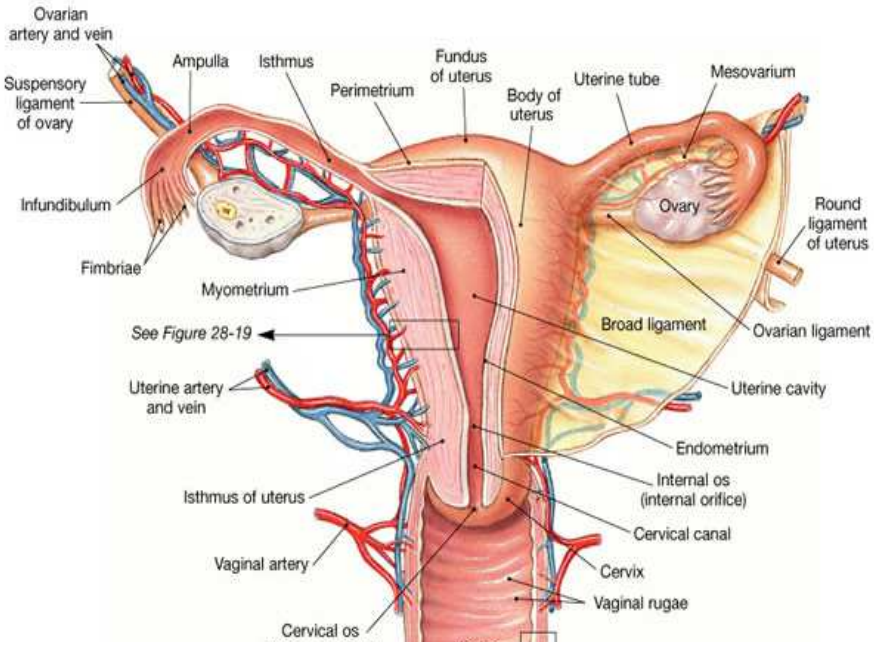


Figure1 Anatomy of the ovary

Vascular supply:

The ovary receives its blood supply from the ovarian arteries. The ovarian arteries arise from abdominal aorta and reaches the mesovarium where it anastomosis with the ovarian branches of internal iliac arteries (Figure 1)

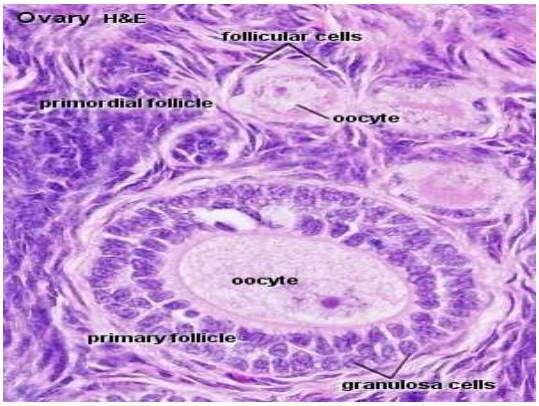
Venous drainage: The right ovarian vein joins the inferior venacava whereas the left ovarian vein enters into the left renal vein.

Lymphatics: It accompanies the arteries and drains into external iliac and para aortic nodes.

Histology:

The ovarian surface is covered by the germinal epithelium which forms the single layer of cuboidal cells. Beneath the epithelium, it has got outer cortex containing follicles and inner medulla containing stroma and hilum. All the follicles are initially primordial in type. During a women's reproductive age group,5-10% of

these primordial follicles will undergo some degree of maturation and changed into primary, secondary, tertiary and Graffian follicles which will releases its oocyte at ovulation[5](Figure 2,3)



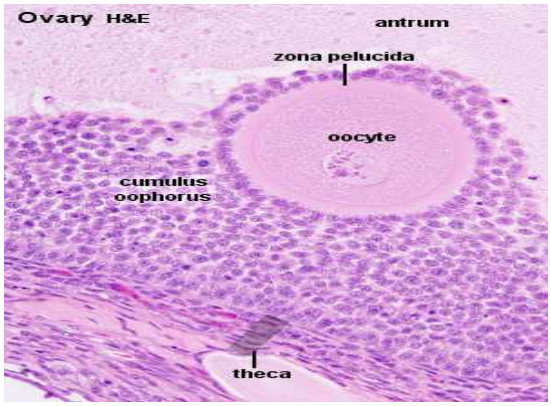


Figure 2 Histology of the ovarian cortex **Figure 3** Histology of Graffian follicle

Functions:

Two main functions of the ovaries are production of ova and hormones; both the functions are controlled through hypothalamo pitutary ovarian axis by endocrine, paracrine and autocrine pathways.

Risk factors of ovarian cancers:

Familial predisposition is noted in 5-10% of cases. Majority are associated with mutation in BRCA1 or BRCA2 and DNA mismatch repair genes. Syndromes associated with ovarian cancers are Lynch II syndrome, Hereditary non polyposis colon cancer syndrome. Individuals with Gonadal dysgenesis are likely to get gonadoblastomas[8].

Risk of ovarian cancer rises sharply after 40 years of age and parallel with gonadotrophin levels. Nulliparity, early menstruation, late menopause, ovarian inflammation are associated with increased risk. One theory says that these events can break the surface epithelium either by monthly ovulation or inflammation; these repeated traumatic insults can be the cause of ovarian cancers. Women with reduced

ovulation by oral contraceptive use, multipara, pregnancy and lactation showed reduced risk[9].

Use of clomiphene citrate, long term estrogen replacement therapy increases the risk of invasive cancers. Dietary factors can also play a role-cheese and meat intake increases the risk whereas higher tea and tomato consumption reduces the risk[9].

Clinical features:

Symptoms of ovarian cancers may not be present until the late stage, afterwards abdominal pain, abdominal mass, and ascites can develop. Precocious puberty and menstrual irregularities were seen in functional ovarian tumours. For early detection of ovarian cancers, serum biomarker assay, transvaginal ultrasonography and genetic studies can be useful, especially in high risk individuals[10].

Spread and metastases

The most common metastatic sites of the ovarian surface epithelial carcinomas and germ cell tumours are contralateral ovary, peritoneal cavity, para-aortic lymph nodes and liver. With intra-abdominal spread, ascites and omental deposits are seen. Mucinous and endometrioid tumors have less tendency to early and widespread peritoneal involvement. Yolk sac tumour and choriocarcinoma are commonly metastatic to lung by haematogenous route[8].

Classification:

The main purpose of classification of ovarian neoplasm is to facilitate the communication between different workers in the field of ovarian oncology be they pathologists, surgeons, epidemiologists or radiotherapists.

In 1967, Novak formulated a classification primarily based on two factors benign or malignant and cystic or solid. It has the advantage of being simple but the borderline tumours fall into the grey zone.[11]

In 1973, World health organization (WHO) formulated a classification based primarily on histogenesis. This classification system was updated in 1999 and subsequently in 2003.[12]

I. SURFACE EPITHELIAL – STROMAL TUMORS

The generally accepted view is that most of these neoplasms arise either directly or indirectly from the surface epithelium, or serosa of the ovary. It accounts for about 60% of ovarian neoplasm and 80-90% of primary ovarian malignancies. Mean age of benign epithelial tumours is 40yrs but malignant epithelial tumours are common in perimenopausal age group[3,8].

Molecular pathogenesis of surface epithelial tumours:

Surface epithelial tumours are most commonly associated with genetic mutations. Recent genetic studies signify that each of these histologic subtypes is associated with distinct molecular genetic variations. High-grade serous and possibly endometrioid carcinomas most possibly arise from invagination of surface epithelium(inclusion cyst) with p53,BRCA1 and/or BRCA2 mutations. Low-grade serous carcinomas probably arise by activation of the mutated RAS–RAF signalling pathway in a stepwise fashion in an adenoma–borderline neoplasm–carcinoma sequence. Mucinous carcinomas are associated with mutations in K-ras oncogene; low-grade endometrioid carcinomas are associated with endometriosis via mutations in CTNNB1 (the gene encoding Beta catenin) and PTEN[13].

The most important group of neoplasms, which arises from epithelial tumours, are classified according to cell type as serous/mucinous/endometroid or clear cell, according to pattern of growth as cystic or solid, according to atypia and invasiveness as benign/borderline/malignant tumours.

A) SEROUS TUMORS:

Architectural and cytological features of serous tumours resemble that of tubal epithelium, this presumably arising as a result of mullerian differentiation along a salphingeal pathway[5].

These tumours comprise 30%of all ovarian tumors. Out of which 70% are benign, 5-10% are borderline and 20-25% are malignant. They are more common in 4-5th decade of life. Bilateral tumours are seen in 12-20% of cases.[3, 8]

Benign serous tumours:

Three basic patterns of these tumours are serous cystadenoma, papillary serous cystadenoma and serous adenofibroma. Serous cystadenomas are predominantly cystic with smooth inner surface, whereas papillary cystadenoma shows papillary excrescences in the inner surface. It may be confused with rare rete cystadenomas but the latter have the hilar location, surrounded by the layer of smooth muscle and exhibit shallow crevices along the inner surface. Serous papilloma appears as polypoidal excrescences on the outer surface.

Third pattern is solid fibromatous tumours, they appear as thin walled cysts containing polypoidal excrescence embedded in a fibrous stroma. Surface papillary adenofibroma should be distinguished from surface stromal proliferations which do not form a mass lesion[5, 8].

Serous borderline ovarian tumours:

They are also called as Atypical proliferating tumour or Serous tumours of low malignant potential.

The mean age of incidence is slightly higher than benign tumours. They are bilateral in 26-36% of cases. In borderline category, at least 10% of the areas showed epithelial stratification and complex stromal papillae. These tumours are separated from carcinoma by the absence of destructive stromal invasion[14.]

Gross appearance is similar to that of the benign tumours but, inner surface showing more friable and more exuberant papillary excrescences. Cystadenofibroma of borderline malignancy has solid, tough, rubbery areas varying from white to yellow in colour.

Microscopically the most characteristic feature of borderline serous tumours is a complex hierarchical branching papillary pattern of growth. The papillae are lined by stratified epithelial cells with nuclear atypia.

Peritoneal implants:

The serous borderline tumours are associated with implants in 30% of cases, which are divided into non-invasive and invasive types. An invasive type shows invasion into the underlying normal tissue as micro papillary structure or solid nests surrounded by clear space. Non-invasive implants are implants with or without stroma, which lack the above features.

Benign bilateral borderline tumours occur in the child bearing age group hence surgery with conservation of as much as ovarian tissue with close clinical follow up until after childbearing is necessary. The overall prognosis remains good even in the presence of implants as they are usually behave as non invasive.[14, 15]

Malignant serous tumours:

These are the most common malignant ovarian neoplasms accounting for approximately 40-50%[16].These are usually large, often bilateral and exhibit a mixture of cystic papillary and solid growth patterns and the surface shows a growth on capsule due to capsular invasion; areas of haemorrhage and necrosis are also seen. Microscopically, well-differentiated type shows clearly formed papillary structures and with an immature connective tissue core. Psammoma bodies are very common in this type. A rare type of low grade serous carcinoma with a favourable prognosis is the psammoma carcinoma in which 75%of the tumour shows psammoma bodies. Moderately differentiated type shows finer and crowded papillae arranged in a lacy like pattern without fibro vascular core. Poorly differentiated serous carcinomas are the most common type which shows loss of micro papillary pattern of growth and the tumour cells are arranged in solid sheets. The cells at the margins are not palisaded and these tumours are associated with syncytial aggregates of tumour gaint cells. Immunohistochemistry plays a very important role in the diagnosis of these serous tumours.

Although serous carcinomas are most often associated with papillae, other carcinomas like transitional cell carcinoma, endometrioid carcinoma, clear cell carcinoma are usually papillary as well, thus entering the differential diagnosis. Endometrioid carcinomas have the villous papillae and associated with focal squamous differentiation which are not seen in serous carcinomas. In clear cell carcinoma, papillae are lined by hobnail or clear cells and typically have hyalinised cores. Transitional carcinomas show broad papillae lined by transitional cells forming undulating thick bands. Recommended strategies for serous carcinomas include presence of precursor lesions like borderline tumours, immunostaining of CK 7

positivity, CK 20 negativity, WT1 and P53 expression. Patients with poorly differentiated carcinomas present with advanced, inoperable stage with a poor prognosis[5, 8].

B) MUCINOUS TUMORS

These tumours have an incidence of 14% of all ovarian tumours and 30.8% of all surface epithelial tumours with a prevalence of 75% being benign, 20% being borderline and 5% being invasive carcinomas. They occur in reproductive age group and are rare before puberty and after menopause. Usually, these tumours are unilateral, only5% of them shows bilaterality[8, 16].

Benign mucinous tumours:

Macroscopically, most of these tumours are usually large with smooth outer surface, the cut surface shows multiloculated cysts containing watery or viscous mucoid material. Microscopically, these are classified as intestinal type and endocervical type depending on the lining epithelium of the cysts. In case of intestinal type, the cyst wall is lined by goblet cells and paneth cells showing picket fence appearance. Endocervical type shows ciliated columnar epithelial lining with abundant intracellular mucin resembling endocervical epithelium.

Borderline mucinous cystadenoma:

These are characterized by stratified cystic lining, not more than 3 layers and may form filiform intracystic papillae with at least minimal stromal support. Nuclear atypia, mitotic figures are seen. Stromal microinvasion is more common than serous tumours. The stromal microinvasion is defined as presence of isolated cells or clusters, each cluster composed of 5-10cells without stromal reaction[15].

Malignant mucinous ovarian tumour (Mucinous adenocarcinoma):

These are relatively uncommon tumours, seen between 40 to 80 years of age group. Macroscopically they are usually large tumours with smooth external surface, cut surface shows closely packed multilocular or thick walled unilocular cyst with an average diameter of 12 to 20cms. It also shows both solid and papillary cystic areas with areas of haemorrhage and necrosis. These are commonly unilateral, bilateral presentation always require exclusion of non-ovarian origin[17].

Microscopically, they show solid growth with loss of glandular architecture, the lining epithelium shows stratification of more than 4 layers and atypical changes with areas of necrosis and stromal invasion. Tumour invasion is ascertained by glands of varying sizes infiltrated in a haphazard pattern or confluent glandular growth with crowded glands that merges together or clusters of single cells with abundant eosinophilic cytoplasm surrounded by clear spaces[5,8]. Mucinous adenocarcinoma should be differentiated from metastatic colon adenocarcinoma. The latter shows bilaterality, surface involvement, nodular growth pattern, lymphovascular invasion, CK 20 positivity and CK 7 negativity[17].

Pseudomyxoma Peritonei:

This entity is associated with mucinous ovarian neoplasm characterized by ovarian tumor with extensive mucinous ascites, cystic epithelial implants on peritoneal surface and produces adhesion. Most of the present studies confirmed that these are the metastases from appendiceal origin[17].

Mixed or collision tumour:

They are defined by the exhibition of two or more of the epithelial elements present in various proportions. It may be a result of biphasic or multiphasic differentiation within the neoplastic tissue. It may also occur with germ cell or sex-

cord stromal tumours. Most commonly, benign mucinous tumours are associated with mature cystic teratoma[5].

C) ENDOMETROID TUMORS:

These tumours account for approximately 20-25% of all ovarian cancers excluding endometriosis, which is considered as non-neoplastic. Usually occur in fifth decade. Most of the endometroid tumors are carcinomas. Less commonly benign forms and borderline cystadenofibroma are encountered. Atypical changes such as atypical hyperplasia within foci of endometriosis and hyperestrogenic state have increased malignant potential to become endometrioid carcinomas. Its coexistence with endometriosis suggests that the origin of tumour directly from ovarian coelomic epithelium[18]. It has a very good prognosis.

Macroscopically, it presents as solid or cystic mass contains friable soft papillae admixed with hemorrhagic content partially filling the cystic lumen. Microscopically, they resemble that of ordinary type of well differentiated endometrial adenocarcinoma. This should be differentiated from metastases uterine endometrial carcinoma. Endometroid carcinoma of ovarian origin may have a foci of endometriosis, secretory change, expansile invasion, squamous metaplasia, and positivity of beta catenin and EMA [5,8,12].

D) CLEAR CELL (MESONEPHROID) TUMORS:

Clear cell carcinoma accounts for about 5% of all ovarian cancers. These tumours occur in 5th to 6th decade of life. Two third of ovarian tumours occurred in nulliparous women are associated with clear cell carcinomas[5].These tumours frequently accompanied by pelvic and ovarian endometriosis[18].Most are carcinomas, benign and borderline ovarian clear cell tumours are very rare. As benign and borderline tumours coexist with clear cell carcinoma, generous sampling is needed.

Macroscopically, they have thick walled unilocular cysts into which projects several fleshy nodules or presents with spongy cut surface. Microscopically the tumour cells are arranged in cystic tubular, papillary and solid sheets. Cores of papillae often exhibits prominent hyalinization lined by one or two layers of polygonal or hobnail or flattened cells. The cytoplasm is clear and often contains glycogen. Extra cellular mucin may be present. Nuclear atypia and mitotic activity is minimal. It should be differentiated from dysgerminoma by its polygonal shape, inconspicuous nucleoli, presence of luminal mucin and EMA positivity[5,8,12].Metastatic renal cell carcinoma is almost indistinguishable, immunomarkers may be helpful in difficult cases, clear cell carcinoma shows CK7positivity and CD10 negativity, whereas the reverse findings are usually present in renal cell carcinoma[17].

E) BRENNER TUMOR: (TRANSITIONAL CELL TUMOR)

These tumours constitute 1 to 2 % of all ovarian neoplasms. The mean age of incidence is 50 years. 5-7% of the tumours are bilateral[16].These are hormonally active and produces hyperestrogenic state due to which uterine bleeding from endometrial hyperplasia occurs. Most authors currently favour an origin from surface ovarian epithelium or cyst derived from them through the process of metaplasia. They can be associated with transitional cell tumour of the bladder and mucinous cystadenoma. Most Brenner tumours are benign out of which 6% are bilateral. Borderline and malignant change is seen in more than 5% in clinically detectable cases.

Macroscopically these are well circumscribed tumours, cut surface shows solid, firm nodule which is whitish or yellowish white in colour varying in size from less than 1cm to massive up to 22cmsto 30cms. Microscopically it consists of solid and cystic nests of epithelial cells resembling the urothelium surrounded by abundant dense fibroblastic stroma. The tumour cells are showing oval nuclei with a

longitudinal groove (coffee bean) and clear cytoplasm. Cystic change may be prominent, in some cases these are lined by mucinous epithelium.

Borderline tumours show more prominent cystic component with papillary lining which consists of 25-50 layers, small spindle to oval shaped atypical cells without mitotic activity. Brenner tumour with irregular infiltrative stromal invasion called as malignant Brenner tumour[5, 8, and 12].

F) TRANSITIONAL CELL CARCINOMA:

These tumours in ovary are broadly similar to transitional cell carcinoma of urinary tract in architectural arrangement and character. Mean age at presentation is around50 to 58 years. Macroscopically the tumours are usually cystic with a few solid areas and a mean diameter of 10 to 30cms.Cut surface shows the cysts with shaggy lining and contain friable polypoidal masses projecting from the walls and exude watery or mucinous fluid with areas of haemorrhage and necrosis. Microscopically the tumour is characterized by tufted papillary growth, slit like glands lined by transitional epithelium with more pleomorphism and mitotic activity without the Brenner component. It has got a good prognosis. It should be differentiated from metastatic tumours from urinary tract. Transitional carcinomas of urothelial origin show CK 20positivity which is negative in ovarian origin[17].

II. SEX CORD STROMAL TUMORS

These comprise a heterogeneous group of neoplasms accounting for 5% of all ovarian neoplasms. It includes those tumours originating ultimately from the sex-cord, mesenchyme or both of the embryonic gonads. They can differentiate either in the direction of testicular type of cells(Sertoli-Leydig cell tumours) or ovarian type(Granulosa cell tumours).They are functional tumours. Inhibin and Calretinin is a

sensitive and specific immunomarker of wide range of sex cord stromal tumors respectively and is of value in differential diagnosis of ovarian neoplasia[5, 8, and 17].

A) GRANULOSA STROMAL CELL TUMOR:

These comprise of two groups:

1) The granulosa cell tumour
2) The thecoma fibroma group

These are the ovarian neoplasms showing differentiation towards follicular granulosa cells. They account for 1-2% of all ovarian tumours and more than 70% of the sexcord stromal tumors. Majority occurs during childbearing age group, less than 5% of the tumours appear before puberty; these are called as juvenile granulose celltumor[19].

a) Juvenile granulosa cell tumour:

It is diagnosed in 80% of cases during the adolescent period presenting with precocious puberty. It can be associated with Ollier's disease or Mafucci's syndrome in a few cases[20].

Macroscopically, these present as large tumours with lobulated outer surface. Cut surface shows solid-cystic portions. The solid areas are yellow or tan in colour and soft in consistency. Microscopically they are showing diffuse cellular growth pattern punctuated by macro follicles and follicle like spaces of varying size and shapes. Neoplastic granulosa cells have abundant vacuolated cytoplasm and lack nuclear grooves admixed with numerous mitotic figures. Juvenile granulosa cell tumour shows very aggressive course and metastasize distantly when compared to adult granulosa cell tumour.

b) Adult granulosa cell tumour:

These are diagnosed during childbearing age group and about 40% of them occur after menopause. Three fourths are associated with hyperestrogenism, which leads to abnormal uterine bleeding. The additional feature of these tumours lies in the fact that all are potentially malignant. But it is difficult from the histological evaluation of granulosa cell tumour to predict their prognostic significance.

Macroscopically, these tumours vary in size with average of 12cms in diameter. Cut section shows solid and cystic areas, solid areas are yellow to greyish white in colour admixed with thin walled cysts filled with haemorrhagic fluid. Microscopically the growth pattern is highly variable. Micro follicular (Call Exner bodies) macro follicular, trabecular, insular, watered silk, solid and diffuse sarcomatoid patterns are seen. Previous studies showed that these patterns indicate about their prognosis like insular and trabecular pattern have good prognosis when compared to sarcomatoid pattern but the recent studies stated that microscopic patterns has no prognostic value, only tumour size and mitotic index have the prognostic significance.[5, 21] An important diagnostic point of this tumour is the presence of folds or grooves in the nuclei resulting in coffee bean appearance. The important differential diagnosis is poorly differentiated carcinomas which shows bilateral involvement, often have spread beyond the ovary, and microscopically shows pleomorphic nuclei with extensive areas of necrosis. Immunohistochemical study of Inhibin positivity adjunct to morphology is helpful in diagnosis of granulosa stromal cell tumor[17].

THECOMA- FIBROMA GROUP:

a) Thecoma:

They constitute 4% of all ovarian tumours. They occur at an average of 53years and can produce estrogenic manifestation. 65%of patients are in the post menopausal age group.[3]Macroscopically; these are unilateral tumours, cut surface shows uniformly solid appearance with well-defined capsule, grey tan to golden yellow in colour, firm in consistency. Microscopically these are cellular tumours composed of fascicles of plump spindle shaped cells. The nuclei are fusiform and have finely dispersed chromatin with lightly eosinophilic or clear or vacuolated the cytoplasm. Special stains like Oil red O demonstrates intra cytoplasmic neutral fat, silver stains demonstrate reticulin around each tumour cells as opposed to granulosa cell tumour in which reticulin surrounds clusters of cells[22].

b) Fibroma:

Fibroma is a neoplasm of unspecialized ovarian stroma. One of the common ovarian tumour occurring invariably after puberty, which is often asymptomatic and discovered incidentally during surgery. Ovarian fibroma can be associated with ascites and right sided pleural effusion (Meigs syndrome).In Gorlin's syndrome they present bilaterally[23].Cellular fibromas may be associated with peritoneal implants.

Macroscopically, these are unilateral tumours with an average of 6cms in diameter. Cut surface shows solid, lobulated appearance, white in colour, firm in consistency. Microscopically they show spindle shaped fibroblastic cells that grow in interlacing fascicles and whorls admixed with collagen. The tumour cells have bland fusiform nuclei with pointed ends.

C) STROMAL TUMOR WITH MINOR SEX CORD ELEMENTS:

These are rare fibro thecomatous tumour containing scattered sex cord elements. This category is only appropriate when the sex-cord component is less than 5% of the tumour. They occur in any age group and are usually hormonally active. Macroscopically, the tumour is solid and indistinguishable from thecoma or fibroma. Microscopically it shows the typical features of thecoma or fibroma in which sex cord structures are inter mingled with the fibro thecomatous cells, sex cord components vary in appearance between fully differentiated granulosa cells and indifferent tubular structures resembling sertoli cells[5].

D) SCLEROSING STROMAL TUMOR OF OVARY:

These are uncommon benign neoplasms occurring in patients between 14-29years of age. The tumour with rare exceptions is hormonally active.

Macroscopically, these tumours arrange from 1 cm to 20cm in diameter, unilateral, cut surface shows solid, lobulated appearance, grey white in colour with occasional yellow foci, firm in consistency. On microscopically examination, they show pseudo lobular pattern of growth, composed of dual cell population:-collagen-producing spindle cells, lipid containing round cells admixed with richly vascularised areas sharply demarcated from hypo cellular oedematous and sclerotic fibrous stroma. Reticulin stain shows the presence of reticulin fibres encircling individual tumor cells. The main differential diagnosis is massive oedema of ovary. In sclerosing stromal tumour, normal ovarian structure displaced by the neoplasm whereas in massive edema of ovary, the normal ovarian structure present within the oedematous areas[5, 8].

E) SERTOLI STROMAL CELL TUMOR:

These are a group of tumours composed of varying proportion of Sertoli cells, Leydig cells and stromal elements which ranges from well to poorly differentiated.

Sertoli Leydig cell tumour:

These tumours as the name indicates are composed of variable mixture of morphologically resembling male Sertoli cells and Leydig cells. These are uncommon tumours, comprise less than 0.1% of ovarian neoplasms[3].Average age of presentation is25yearsandclinically presents with features of defeminisation and musculinisation. This tumour was previously called as Arrhenoblastoma or Androblastoma.

Majority of the tumours are unilateral, firm, yellow to tan, solid masses with smooth external surface. The microscopic pattern is extremely variable. Based on the amount of hollow tubules, variable types like well differentiated or intermediate or poorly differentiated arrhenoblastoma exists. About 20% of the tumours show heterologous elements, most commonly it shows mucinous epithelium of gastrointestinal tract, other components like skeletal muscle, cartilage and neuroendocrine cells have also been described. Immunohistochemically testosterone and estradiol are found in both Sertoli and Leydig cells[24]. The area of sertoli cell differentiation stain for Keratin and gonadal stromal component is positive for Inhibin[17].The prognosis of Sertoli -Leydig cell tumour is good which correlates with the stage and degree of differentiation of the tumor[8].

F) SEX CORD TUMOR WITH ANNULAR TUBULES:

It is a distinctive ovarian tumour, which occurs in two forms: as a multifocal bilateral tumours with the Peutz-Jeghers syndrome or as a solitary neoplasm without the syndrome.50% of patients present with hyperestrogenism. In several tumours the pattern merged with that of granulosa cell tumour admixed with elongated solid tubules resembling Sertoli cells. The characteristic microscopic feature is the presence of complex annular tubules filled with eosinophilic material, hyaline bodies associated with calcification. Syndrome associated tumours are typically showing

bilaterality, calcification and swollen appearance. The microscopic appearance is similar to that of gonadoblastoma from which it differ only by clinical and genetic background[5, 8].

G) GYNANDROBLASTOMA:

Rarely mixtures of Granulosa cell tumours and Sertoli-Leydig cell tumours have been reported. So both androgenic and estrogenic manifestations produced by the same neoplasms. To make this diagnosis well differentiated elements of both the tumours should be present in equal amounts[8, 12.]

H) LIPID CELL (LIPOID, STEROID CELL) TUMOR

The exact origin of this tumour remains undecided. Therefore more recently the term steroid cell tumour has been proposed for the entire group, with added designation of Leydig cell type and cortical type whenever necessary. These tumours account for 0.5% of all ovarian neoplasms. It can occur at any age group and are associated with virilising syndrome and defeminisation.

Macroscopically, these are usually unilateral, well circumscribed and are composed of yellowish nodules separated by fibrous septa. Microscopically these are characterized by masses of large polyhedral cells with abundant eosinophilic or vacuolated cytoplasm that shows positivity for fat stains. About 25% of tumours have tendency to develop malignancy. Malignant tumours tend to be larger with foci of necrosis and haemorrhage. Nuclear atypia and mitotic activity also seen[5, 8].

III.GERM CELL TUMORS:

These tumours constitute approximately 20%of all ovarian neoplasms, out of which 3% are malignant tumors. Germ cell tumours are the commonest ovarian neoplasm in the first two decades of life constituting approximately two third of all ovarian tumors. The younger the age, the occurrence of malignancy will be high[3, 25].

95% of the germ cell tumours are benign cystic teratoma[25]. The current pathogenetic theory suggests that, these tumours arising from the germ cell which has undergone defective meiotic division[8].Most of the malignant ovarian tumours occur in pure form; approximately about 8% are composed of two or more subtypes called as malignant mixed germ cell tumour.

A) DYSGERMINOMA:

These are the most common germ cell tumours of the ovary accounting for 1% of all ovarian cancers and for 5-10% of ovarian cancers in first two decades of life.10% of the tumours are bilateral. It is also the most common malignant tumour, which occurs in patients with gonadal dysgenesis[8, 25]. Most commonly they present with abdominal mass and pain. Macroscopically, these are solid tumours with an average diameter of 15 cms and have a smooth bosselated external surface. Cut section shows a typically solid, lobulated, fleshy appearance; tan or white in colour and soft in consistency. Microscopically, the tumour cells are arranged in nests separated by fibrous septa with lymphocytic infiltration. Germinoma cells are round cells with abundant clear to eosinophilic cytoplasm, prominent cytoplasmic membranes, round vesicular nucleus and central prominent nucleoli. Periodic Acid Schiff stain helps in demonstrating the presence of intra cytoplasmic glycogen.

Anaplastic dysgerminoma: This is a variant of dysgerminoma where the tumour cells are arranged in pseudo glandular or tubular pattern with a high mitotic rate, accompanied high nuclear atypia which simulates the embryonal carcinoma. Immunostainig of these tumours show a strong positivity usually a membranous pattern for Placental alkaline phosphatase and CD117.Especially CD117 is useful in differentiating the two, because embryonal carcinoma is CD117 negative.[8, 26]

B) YOLK SAC TUMOR (ENDODERMAL SINUS TUMOR):

They are almost as common as dysgerminoma in younger women, constituting 20% of primitive germ cell tumour. Most of the patients present with abdominal pain, distension or abdominal mass.

Macroscopically, these tumours tend to be unilateral and large with an average diameter of 16 cms. Cut section shows a soft, friable, and yellow to greyish white tissue often containing cysts with extensive areas of haemorrhage and necrosis. Microscopically these tumours have a more than one pattern, which could be reticular, solid, festoon, pseudo papillary, and hepatoid or polyvesicular vitelline pattern. Schiller Duval bodies are the characteristic feature of this tumour. They consist of papillae lined by tumour cells that project into dilated cystic spaces, resulting in somewhat glomeruloid appearance. Eosinophilic hyaline globules which are PAS positive diastase resistant are seen in yolk sac tumors[5, 8.]Small enteric glands are lined by columnar or goblet cells or malignant embryonal stroma containing spindle or satellite cells are present in yolk sac tumors. They consistently produce alpha fetoprotein which can be demonstrated in tissue sections by immunohistochemistry or in serum. They also immunoreactive for Cytokeratin, Alpha1 antitrypsin but not with EMA [17.]

Clear cell carcinoma is the tumour more often confused with Yolk sac tumor. The nuclei in Yolk sac tumours are almost always more primitive in appearance and the papillae are simple containing a single central vessel and lacking the hyalinised core in contrast to clear cell carcinomas. Immunoprofile of CK7 positivity, EMA positivity and Alpha fetoprotein negativity strongly favours the diagnosis of clear cell carcinoma[27].

C) EMBRYONAL CARCINOMA:

Embryonal carcinoma is very rare accounting for at most 3% of primitive ovarian germ cell tumors. These tumours occur in younger age group with a median age of 15 years presenting with precocious puberty, vaginal bleeding, hirsutism and amenorrhea. Macroscopically, it shows smooth and glistening external surface with a mean diameter of 17cms.Cut surfaces are predominantly solid and variegated with areas of haemorrhage and necrosis. Microscopically it shows solid sheets and nests of tumour cells, which are large primitive cells with occasional papillae and abortive glandular structures. Isolated syncytiotrophoblastic gaint cells are always present. Serum alpha-fetoprotein is often elevated whereas chorionic gonodotropin are invariably high to a level that results in consistently positive pregnancy tests[5, 8,12.]These tumours may be confused with a poorly differentiated adenocarcinoma or undifferentiated carcinoma, however such tumours typically occur in the late reproductive or postmenopausal age group, rarely elaborate alpha fetoprotein and they lack trophoblastic differentiation[27].

D) POLYEMBRYOMAS:

Embryonal carcinomas consists of numerous embryonal bodies are called as Polyembryomas. These structures are highly organised, showing an embryonic disc, yolk sac and amniotic cavities[5].

E) CHORIOCARCINOMA:

Primary ovarian choriocarcinomas account for less than 1% of primitive ovarian germ cell tumours. Most Choriocarcinomas are as a result of metastasis from uterine tumors. So some authors using the strict criteria would only accept as non-gestational Choriocarcinoma those cases occurring before puberty, because ovarian gestational choriocarcinoma have a better prognosis than non-gestational

choriocarcinoma[5].Pure choriocarcinomas are typically solid, haemorrhagic and friable masses. Microscopically they show typical biphasic plexiform pattern consisting of admixture of syncytial and cytotrophoblastic elements in a necrotic and hemorrhagic background.

Immunohistochemical reactivity for HCG (human chorionic gonodotropin) is the rule in addition keratin7 is said to represent a marker for subtype of trophoblastic cells. Typical microscopic picture with immunomarkers used to distinguishes from exceptionally rare ovarian carcinoma of surface epithelial origin exhibiting choriocarcinomatous differentiation[27].

F) TERATOMA:

Teratomas are composed of tissues representing at least two, but usually all three embryonic layers. They are divided into three types- Immature teratoma, mature teratoma and Monodermal teratoma.

1) IMMMATURE TERATOMA:

It is a malignant teratoma in which the malignant elements have an embryonal appearance. These are the third most common primitive germ cell tumour accounting for almost 20% of all cases and 1% of ovarian cancers in general[25].It occurs in children and young adult with an average age of presentation is 20 years. These are unilateral tumours with average diameter of 17 to 18 cms. Cut section shows partly solid and cystic areas, solid areas are typically soft, fleshy with focal haemorrhage and necrosis.

Microscopically it shows the tissues derived from all the three germ layers, growing as disorganized mixture of mature and immature elements. Neuroepithelial tubules and rosettes are easily recognized elements that are indicative of immaturity in

teratoma. Immature cartilage, fat, endodermal glands, and liver tissue are also recognized.

Table 1 Grading of immature teratoma

Grade	Immature tissue	Amount of Neuro epithelium
1	+	Rare , not > 1 LPF / slide
2	++	Common, not > 3 /LPF / slide
3	+++	Prominent ,>4/LPF/slide

Immature teratoma must be graded, since the prognosis depends on the grade as well as the stage. The most widely used grading system (Norris system). Norris et al stratifies immature teratoma into three grades based on the amount of immature neuroepithelial elements[28] (Table1)

Grade 3 tumours can be associated with undifferentiated cells with occasional differentiations into glial masses, ependymal, neuroblastic and medulloepithelioma like arrangements[5].Serum Alfa fetoprotein is a useful marker in this tumour as some patients have shown increased levels. Immature teratoma should be differentiated from carcinosarcoma with heterologous elements; the former shows neuroepithelial elements and occur in younger age group.

2) MATURE CYSTIC TERATOMA:

The most common benign tumour in Western world is benign mature cystic teratoma or dermoid cyst, account for approximately 25% of all ovarian tumors, 30% of benign ovarian tumors; 85-90% of germ cell tumor.85% of patients is between 20 to 50 years of age. It is unilateral in 88%of cases.[24]

Macroscopically, they are multiloculated cysts, with a greasy content largely composed of keratin, sebum hair and teeth. Sometimes the tumour contains imperfectly formed mandible or even partial human body like

configuration(homunculus) called as fetiform teratoma. Single or several rounded, polypoidal masses designate Rokitansky protuberances and composed of variety of tissue types. If solid areas are present it should be sampled carefully to rule out immature teratoma. Microscopically the tumour contains a variable admixture of ectodermal, mesodermal, and endodermal elements such as hair follicles, epithelium, salivary gland, thyroid, and respiratory tract epithelium. Rarely a benign cystic teratoma is associated with peritoneal glial implants or peritoneal melanosis. Mature cystic teratoma can coexist with mucinous cystadenoma, Brenner tumour and fibrothecoma. Secondary malignancy can occur in benign cystic teratoma; this complication is common in postmenopausal women. Secondary neoplasms are usually unilateral, but contralateral ovary may contain a benign cystic teratoma. A variety of benign and malignant tumours can occur; benign tumours include cutaneous adnexal tumour and benign salivary gland tumours, whereas the most common malignant tumour is squamous cell carcinoma[29.]

3) MONODERMAL TERATOMA:

This term applies only to the unilateral differentiation of tissues derived from one of the germ cell layers. Struma ovary is the most common tumour in this group; carcinoid tumour and neuroectodermal tumours are next commonly encountered tumours.

a) STRUMA OVARII:

Thyroid tissue is the unique and predominant component in this teratoma. Most are benign and malignant Struma ovary is very rare. The features that define malignancy include invasion of one cell into another or papillary pattern with typical nuclear characteristics. Macroscopically, these tumours are less than 10cm in diameter Cut section shows solid, tan, or brown in colour with a glistening appearance.

Microscopy shows the tumour consists of thyroid follicles filled with eosinophilic colloid and lined by columnar or cuboidal bland cells[5, 8].

b) CARCINOID TUMOR:

These neuroendocrine tumour occurs in older age group, presenting with menstrual irregularities and pelvic and abdominal pain. Macroscopically, the primary carcinoids are unilateral, cut surface shows solid tumour, tan or yellow in colour. Microscopical examination shows the tumour cells composed of uniform round to cuboidal cells arranged in trabecular pattern that have a round nucleus with coarse salt and pepper chromatin. The ovary is the most common site of metastasis from carcinoid of gastrointestinal tract (usually from small intestine or appendix)[27].Evidence favouring for metastatic carcinoid includes bilaterality, intra ovarian growth in the form of several nodules, probable presence of a carcinoid in the other sites. Neuroendocrine immunomarkers like Serotonin, Chromogranin shows positivity[5,8.]

G) MALIGNANT MIXED GERM CELL TUMOR:

These comprise 5 to 20 % of all malignant germ cell tumors[3]. These contain a mixture of malignant germ cell elements, commonly shows a combination of dysgerminoma and yolk sac tumour, followed by immature teratoma, Embryonal carcinoma and Choriocarcinoma. They occur exclusively in children and young women with an average age presentation are 16 years. Tumours composed of Yolk sac tumour, or choriocarcinoma, or highly immature teratoma have poor prognosis, than when dysgerminomas or more mature teratomas predominate.[30]

H).MIXED GERM CELL-SEXCORD-STROMAL TUMORS:

A) GONADOBLASTOMA:

These are tumours composed of a combination of germ cells and sex cord stromal cells .It is also known as dysgenetic gonadoma. As per the name, these occur in sexually abnormal individuals affected by gonadal dysgenesis; commonly associated with XY gonadal dysgenesis but not in XX gonadal dysgenesis.30% of the tumours are bilateral and usually are small; become apparent only by microscopic examination. Microscopically these are characterized by admixture of primitive germ cells resembling those of dysgerminoma with sex cord stromal cells resembling that of immature sertoli and granulosa cells. Hyalinization and calcification are common. Gonadoblastoma can be confused with sex cord tumours with annular tubules but the latter lacks the germ cell component[5, 8,12].

IV) TUMORS OF THE RETE OVARII:

These are relatively uncommon tumours, which includes adenoma, cystadenoma and carcinoma.

V) TUMORS OF UNCERTAIN ORIGIN

A) SMALL CELL CARCINOMA:

There are two types of small cell carcinoma designated as hypercalcemic and pulmonary type respectively.

Hypercalcemic type: It is by far the most common form of undifferentiated carcinoma, which occurs in young females and is nearly always bilateral. These tumours are associated with hypercalcemia in two third of cases which disappear following removal of tumor. Macroscopically these tumours are large, solid with areas of haemorrhage and necrosis. On microscopical examination it shows diffuse population of small, closely packed cells of scanty cytoplasm, small hyperchromatic

nuclei containing prominent nucleoli with numerous mitotic figures. Cytoplasmic hyaline globules are seen. The prognosis is very poor because of frequent extra ovarian spread.

Pulmonary type: This tumour resembles in all regards with lung tumour. It may be pure and associated with endometrial carcinoma. Immunohistochemistry reveals these tumours are positive for keratin, EMA, NSE and rarely for chromogranin[17].

B) TUMOR OF PROBABLY WOLFFIAN ORIGIN:

This tumour is now renamed as Wolffian adnexal tumour; because the exact histogenesis is still arguable whether it could be due to mesonephric origin or sex cord stromal origin or it has been regarded as of uncertain origin. These are benign tumours which are grossly either solid or cystic, grey white or yellow in colour. Microscopically, these tumour epithelial cells grow in the form of cystic structures, solid or hollow tubules and diffuse sheets. They show negative staining for mucin. Immunohistochemistry shows positivity for keratin (CK7 but not CK 20) Calretenin and Vimentin. In a few cases Estrogen, Progesterone and Inhibin positivity is also recorded[17].

VI. UNCLASSIFIED TUMORS

SARCOMAS:

The primary sarcoma of ovary is extremely rare. Some of the soft tissue tumours, which occur in ovary, are fibrosarcoma, endometrial sarcoma, leiomyosarcoma, chondrosarcoma, osteosarcoma, rhabdomyosarcoma, angiosarcoma and peripheral neuroectodermal tumour.

Fibrosarcoma:

These are rare ovarian tumours but one of the most common ovarian neoplasms. Commonly occur in postmenopausal age group. Macroscopically they show solid fleshy cut surface with areas of haemorrhage and necrosis. Microscopically these tumours show spindle shaped cells arranged in herringbone pattern with nuclear atypia admixed with numerous mitotic figures[12.]

VII. METASTATIC TUMORS (SECONDARY TUMORS)

The ovary is a common frequent site of metastasis from stomach, large bowel, appendix, breast, uterus, lung and skin. Features of metastasis include bilaterality, surface involvement by tumour cells, infiltrative nodular pattern of growth, ovarian hilar involvement, single cell invasion, presence of signet ring cells and dirty necrosis.[31]

Macroscopically, they vary in appearance; ovary is completely replaced by solid white or tan metastatic tumour or grows as large unilocular or multilocular cystic tumour with solid areas. Bilaterality and multiple serosal implants on ovary are particularly suggestive of metastasis.

Microscopically there is a multifocal involvement of the ovary with implants on the surface. The tumour cells may diffusely infiltrate the parenchyma or grow in discrete nodules. The diagnosis of metastasis is difficult when the secondary disease simulates the primary tumour. Immunohistochemical stains like CK 7 and CK 20 are helpful in establishing the diagnosis, primary ovarian tumours shows CK 7 positivity and CK 20 negativity[17].

KRUKENBERG TUMOR:

It is a form of metastatic cancer in which signet-ring type cell grows in abundant hyper cellular stroma. The stomach is the usual primary site for this tumour.

It shows high incidence in females and Japanese population. Macroscopically the ovaries are usually retaining their shape but they are symmetrically or asymmetrically enlarged. Cut section shows homogenous appearance with firm, yellowish white areas, soft in consistency. Microscopically the tumour cells grow in as isolated single cells, form nests, cords or tubules. The tumour cells contain round cytoplasmic vacuoles that compress and flatten the hyper chromatic nucleus against the one cell border resulting in signet- ring appearance. The stroma shows pools of mucin. Special stains for mucin (PAS, Alcian blue) andimmunostains of epithelial markers are helpful in diagnosis. However according to Novak and Gray criteria, it is diagnosed if presence of intracellular mucin within the neoplastic signet ring cells and diffuse sarcomatoid proliferation of ovarian stroma. Prognosis of Krukenberg tumour is still very poor but there are no established criteria for it[32].

PROGNOSIS OF OVARIAN TUMORS:

The prognosis of ovarian tumours depends on various factors. The important factors influencing the ovarian cancer prognosis are histological type, stage of ovarian cancer, molecular abnormality, woman's age and general health-whether the cancer has just been diagnosed or has recurred. TNM and FIGO Classification of staging is used in ovarian cancers [8, 33].

NON NEOPLASTIC LESIONS

Non neoplastic lesions may form pelvic masses, associated with hormonal manifestations, mostly simulating ovarian neoplasm, so recognizing them is important from the view point of therapy and prognosis.

EPITHELIAL INCLUSION CYST:

These arise from cortical invagination of surface epithelium that has lost their connection with the surface. Those that develop in fetal life is called fetal inclusion

cyst lined by primitive celomic epithelium and those that develop as aging process, lined by celomic epithelium are named as acquired inclusion cyst. They can measure up to 1cm in diameter. Most are incidental findings in the form of single to multiple cysts scattered throughout the superficial cortex. The infrequent finding of dysplastic epithelium lining the cyst support the hypothesis that they give rise to surface epithelial carcinoma.

In few instances hydropic change of the lining epithelial cells can mimic that of signet ring cell carcinoma, the latter shows deep invasiveness, presence of intracellular mucin and can be associated with necrosis[27].

FOLLICULAR CYST:

They develop from distension of atretic or developing follicle and do not exceed 10cm in diameter and when less than 2.5cm are called as cystic follicle. It may occur in any age, infancy to menopause. In women with reproductive age group, it is associated with menstrual irregularities, or may rupture causing acute abdominal pain with haemoperitoneum.

Macroscopically, they are unilocular, smooth surfaced, thin walled cysts containing serous or serosanguinous fluid. Microscopically, they are lined by inner granulosa cells and outer theca interna cells with luteinisation in either layer of cells. Foci of luteinised cells show marked pleomorphism in pregnancy and puerperium. This should be differentiated from cystic granulosa cell tumour which is rarely uniformly luteinised with nuclear grooves and never containing bizarre nuclei. [5, 27]

CORPUS LUTEAL CYST:

They are single and usually less than 6cm in diameter. They usually develop at the end of menstrual cycle or may occur in pregnancy. Macroscopically, the cyst wall is yellow or convoluted, often containing bloody fluid. Microscopically, the cyst wall

is composed of thick convoluted layer of large luteinised granulosa cells interrupted externally by wedges of much smaller theca lutein cells. These architectural and cytological features are important in differentiating from luteinised follicular cyst.[5, 27]

POLYCYSTIC OVARIAN DISEASE, STROMAL HYPERTHECOSIS, STROMAL HYPERPLASIA:

These disorders may be associated with androgenic or estrogenic manifestations.

Polycystic ovarian disease or Stein Levanthal Syndrome:

PCOD has been estimated in about 3.5-7% of female population. Clinical manifestations are obesity, hirsuitism, menstrual irregularities with increased serum luteinising hormone and decreased follicle stimulating hormone. Macroscopically, they are opaque with thickened capsule; cut section revealed equally sized multiple cysts, situated superficially beneath the capsule with central homogenous stroma. On microscopic examination, the outer cortex is hypo cellular, containing cysts lined by hyperplasic, enlarged, lipid-laden theca interna cells. Granulosa cells are less conspicuous and are not luteinised. It is managed medically by regulation of menstrual cycle & ovulation induction[5, 8].

Stromal hyperplasia and stromal hyperthecosis:

They are most commonly present in postmenopausal women at 6^{th} to 7^{th}decade.It may be associated with estrogenic or androgenic effects, obesity, hypertension. Gross features are bilateral ovarian enlargement, each ovary measures approximately 8cm in diameter simulating ovarian tumour. Cut surface shows homogenous appearance, white to yellow in colour, firm in consistency. Microscopically they show diffuse or nodular proliferation of plump ovarian cortical

cells encroaching on the medulla. It may be associated with endometrial hyperplasia, and adenocarcinoma[5,8].

ENDOMETRIOTIC CYST:

Ovary is the most common site of endometriosis[34].Pain associated with menstrual cycle is the most common symptom. Macroscopically, they show small, slightly raised blueberry spots on the ovarian surface, often accompanied by fibrous adhesions and in extensive involvement the entire ovary may be converted into chocolate cyst. On microscopical examination ovary shows endometrial glands and stroma associated with fresh or old haemorrhages. It may be the site of reactive atypia, hyperplasia, metaplasia or malignancy. Endometrioid adenocarcinoma, clear cell carcinoma are thought to arise from foci of endometriosis[18].

MASSIVE EDEMA OF OVARY:

Accumulation of edema fluid within the ovary has been designated as massive ovarian edema. These are common in younger age and unilateral in 90% of the cases. Macroscopically, they present as enlarged, soft, fluctuant ovary with smooth outer surface. Sectioning shows homogenous, pale appearance, exuding a watery fluid. Microscopically it shows abundant pale staining fluid that surrounds the follicle and typically spares the superficial cortex. It should be differentiated from ovarian neoplasm with oedematous and myxoid appearance, such as sclerosing stromal tumour, Krukenberg tumour and edematous fibroma[27].

GRANULOSA CELL AND SERTOLI CELL PROLIFERATIONS:

Focal proliferations of granulosa cells commonly occur during pregnancy. They typically develop in atretic follicles, microscopy reveals small aggregates of granulosa cells that shows trabecular, diffuse, insula rpattern. They are differentiated

from small granulosa cell tumours by their microscopic size, multifocality, confinement to atretic follicle and common occurrence during pregnancy[35].

PREGNANCY LUTEOMA:

It is a tumour derived from luteinised theca and stromal cells. Grossly they appear as bilateral, multiple, solid nodules with soft, fleshy, yellowish brown cut surface. Microscopic examination shows sharply circumscribed nodules of polygonal cells with granular, eosinophilic cytoplasm. This tumour should be differentiated from sex cord stromal tumours, where the latter is almost always unilateral, solitary and have a denser reticulin pattern[27, 35].

INFLAMMATORY LESIONS OF THE OVARY:

Pelvic inflammatory disease of bacterial origin accounts for most common infection in developed countries. Ovarian involvement stems from salpingitis and typically takes in the form of bilateral tubo ovarian abscess and healed abscess is converted into a tub ovarian cyst, that may mimic ovarian cystic neoplasm during surgery. So careful microscopical examination of inflamed tubal plicae gives a clue to the diagnosis.

Ovarian involvement is present in only 10% of cases of pelvic tuberculosis[36].On gross examination the ovaries typically have tubal-ampullary adhesions. Microscopically they show caseating granulomas confined to the ovarian cortex.

Other infectious agents producing granulomatous oophoritis are actinomyces schistosomiasis, whereas non-infectious granulomatous causes are sarcoidosis, foreign body granuloma and Crohn's disease.[37]

MATERIALS AND METHODS

Our study is a retrospective study carried out in the Department of Pathology, Tirunelveli Medical College, Tirunelveli from Januaruy 2008 to December 2010.This study was undertaken to find out the incidence and analyse the histomorphological patterns of ovarian lesions in and around Tirunelveli.

Clinical details of the patients including the age, parity, hormonal status and examination findings were recorded from the case sheets available in the Medical Records Department . The necessary laboratory parameters including serum markers like CA-125, Alpha Feto Protein, Human Chorionic Gonadotropin if done were noted. The reports of all the radiological investigations viz.Ultra sonography, Computerised Tomography, MRI and FNAC report were recorded. The cases with diagnostic difficulties were discussed with the gynaecologists.

Gross and microscopic features of all the cases were noted from the records available in the Department. Slides and blocks were retrieved from the store, further sections were taken from the block and stained with hematoxylin and Eosin and the findings were noted. Special stains like PAS, Reticulin, Alcian blue were performed as per standard staining protocol (Appendix II). Immunohistochemical markers including Cytokeratin, Epithelial membrane antigen, Alpha Inhibin and Alpha feto protein were performed in equivocal cases. Finally histopathological findings of all the cases were analysed and the tumors were classified according to recent WHO classification.[12]

INCLUSION CRITERIA :

1. All patients who are preoperatively diagnosed as having ovarian lesions and operated in Tirunelveli Medical College.

2. Patients of all ages.

3. Both neoplastic and non-neoplastic ovarian lesions were included in this study.

EXCLUSION CRITERIA:

Incidentally found ovarian lesions in patients operated for other gynaecological problems were excluded from this study.

OBSERVATION AND RESULTS

This retrospective study included the period of 3 years from January 2008 to December 2010, in Department of Pathology, Tirunelveli Medical college.203 ovarian lesions were analysed, of these 165 cases were neoplastic lesions,38 cases were non neoplastic lesions.

Clinical features, gross and microscopic features were recorded in a proforma (Appendix I). These details were then transcribed into a master sheet and were analysed.

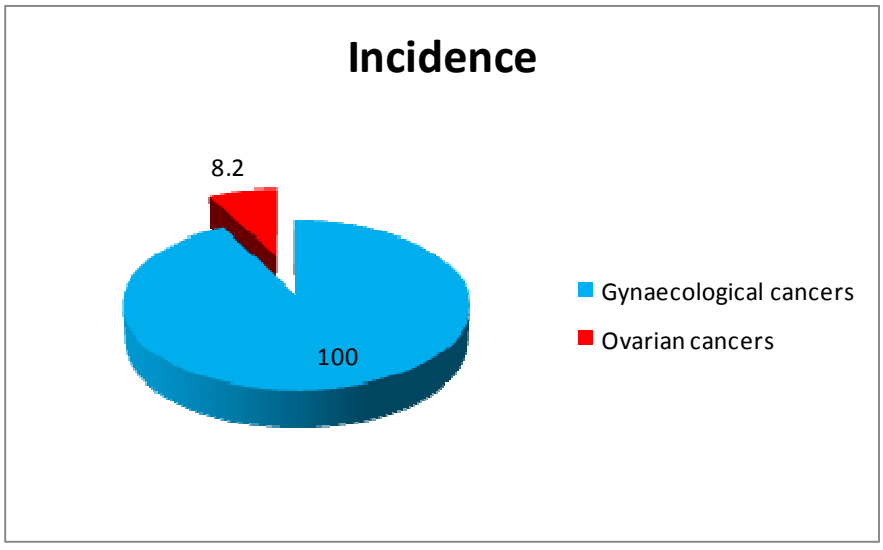
INCIDENCE OF OVARIAN CANCERS:

TABLE 2 Incidence of ovarian cancers among gynaecological cancers

S.NO	Type of Cancers	No of cases	Percentage (%)
1	Gynaecological cancers	439	100
2	Ovarian cancers	36	8.2

As shown in Table 2, during the period of Jan 2008 –Dec 2010, 439 gynaecological cancers were reported, out of which 36 cases were ovarian cancers, accounting for 8.2% incidence of total gynaecological cancers. (Chart 1)

Chart 1 Incidence of ovarian cancers among gynaecological cancers



DISTRIBUTION OF NEOPLASTIC AND NON NEOPLASTIC OVARIAN LESIONS

TABLE 3 Distribution of neoplastic and non-neoplastic lesions.

Non neoplastic lesions		Neoplastic lesions		Total
No of cases	**Percentage(%)**	**No of cases**	**Percentage(%)**	
38	18.71	165	81.28	203(100%)

As shown in Table 3, among 203 ovarian lesions,165 cases were neoplastic lesions, accounting for 81.28%, 38 cases were non neoplastic accounting for 18.71%. Increased incidence of neoplastic lesions was observed in our study.

DISTRIBUTION OF BENIGN, BORDERLINE AND MALIGNANT LESIONS:

TABLE 4 Distribution of benign, borderline and malignant ovarian lesions

Benign tumours		Borderline tumours		Malignant tumours		Total(%)
No of cases	**(%)**	**No of cases**	**(%)**	**No of cases**	**(%)**	
127	76.96	2	1.21	36	21.81	165(100%)

As shown in Table: 4, among 165 neoplastic lesions, 127(76.96%) cases were benign, 36(21.81%) cases were malignant, and 2 (0.01%) cases were borderline tumours. Increased incidence of benign tumours were observed in this study.(Chart 2)

AGE DISTRIBUTION AMONG NEOPLASTIC LESIONS:

TABLE 5 Distribution of benign, borderline and malignant ovarian neoplasm in different age groups

S.No	Age group	Benign tumours		Borderline tumours		Malignant tumours		Total (%)
	Years	No of cases	%	No of cases	%	No of cases	%	
1	<19	3	2.36	0	0	2	5.55	5(3.03)
2	20-39	56	44.09	1	50	6	16.66	63(38.18)
3	40-59	54	42.51	1	50	16	44.44	71(43.03)
4	>60	14	11.02	0	0	12	33.33	26(15.75)
	Total	127	100	2	100	36	100	165(100)

Peak incidence of benign ovarian tumours were in the age group of 20-39years (56cases,44.09% of all benign ovarian neoplasms).Peak incidence of malignant ovarian neoplasm was seen in the age group of 40-59years (16 cases,44.44% of all malignant neoplasms).The youngest patient was 15 year old, whereas the oldest patient was 77 year old.(Table 5)(Chart 3)

Chart 2 Distribution of Benign. Borderline and Malignant neoplasms

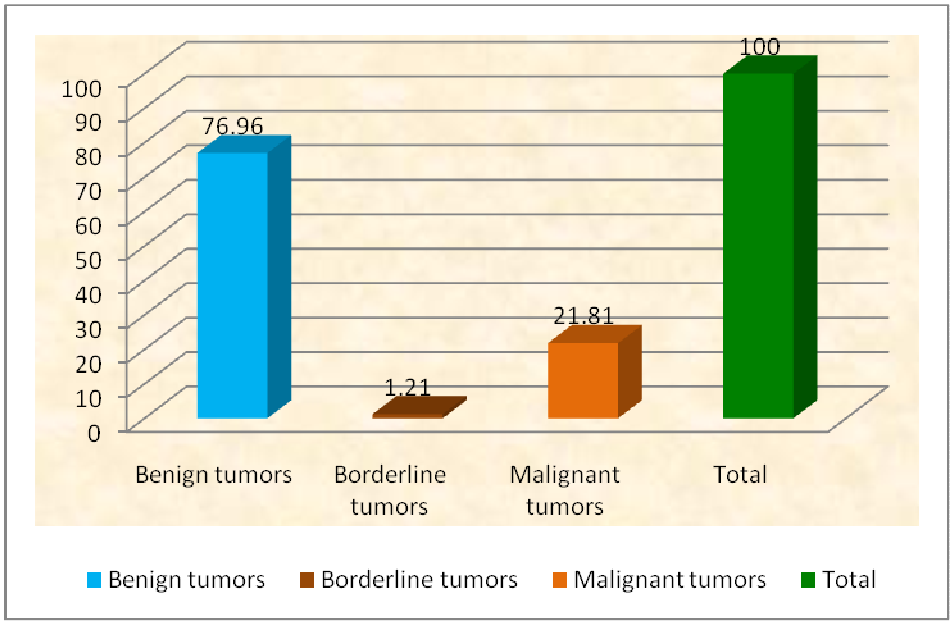
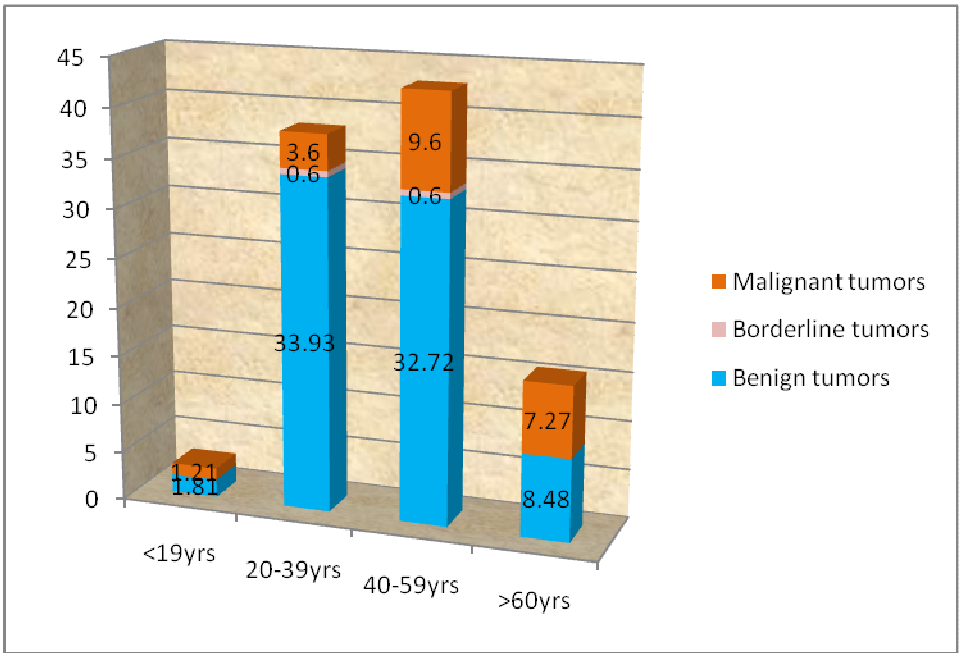


Chart 3 Distribution of benign, borderline and malignant ovarian neoplasm in different age groups



CLINICAL PRESENTATION OF NEOPLASTIC LESIONS:

TABLE 6Mode of presentation of neoplastic lesions

S.No	Clinical presentation	No of cases	%
1	Abdominal pain	52	31.51
2	Abdominal mass	23	13.93
3	Bleeding per vagina	5	3.03
4	Abdominal pain with mass	78	47.27
5	Abdominal pain with bleeding per vagina	7	4.24
	Total	165	100

Among the neoplastic lesions almost half of the cases were associated with combination of symptoms, commonly presented with abdominal pain and abdominal mass (78cases,47.27%). Abdominal pain was the single most presenting symptom followed by abdominal mass (52 cases, 41.51%) (Table 6).

LATERALITY OF THE NEOPLASTIC LESIONS:

TABLE 7 Laterality of Benign, Borderline and Malignant tumours.

S.No	Tumour type	Laterality				Total
		U/L No of cases	%	B/L No of cases	%	No of cases
1	Benign	123	97.63	4	2.36	127
2	Borderline	2	1.21	-	-	2
3	Malignant	22	61.11	14	38.88	36
	Total	147		18		165

In our study, 97.63% of the benign tumours were unilateral whereas 38.88% of the malignant tumours were bilateral. Bilaterality was more common in malignant tumours.(Table 7)

GROSS FEATURES OF NEOPLASTIC LESIONS:

1) TUMOUR SIZE:

TABLE 8 Variation in size among neoplastic lesions

S.No	Size (cms)	No of cases	Percentage (%)
1	0-10	95	57.57
2	11-19	53	32.12
3	20-29	14	08.48
4	30 and above	3	01.81
	Total	165	100

Among the neoplastic lesions majority of the cases were less than 10cms (95 cases; 57.57%). (Table: 9). The smallest tumour had a size of 5x3x3cms, diagnosed as serous cystadenofibroma. Largest tumour measured 35x30x10cms which was diagnosed as mucinous cystadenoma. (Table 8)

2) CONSISTENCY:

TABLE 9 Gross morphology of the ovarian lesions

S.No	Gross morphology	No of cases (%)			Total(%)
		Benign	Malignant	Borderline	
1	Cystic	109(99.09%)	0	1(0.9%)	110(66.6)
2	Solid	6(30%)	14(70%)	0	20(12.12)
3	Solid and cystic	11(31.42%)	23(65.71%)	1(2.8%)	35(21.21)

Present study showed that 99% of the benign tumours were cystic whereas 70% of the malignant tumours were solid in consistency. 65.7% of the malignant tumours had solid-cystic cut surface. (Table 9)

DISTRIBUTION OF OVARIAN NEOPLASMS ACCORDING TO THE HISTOLOGICAL TYPE:

Table 10 Distribution of ovarian neoplasms based on histological types

S.No	Types	No of cases	%
1	Surface epithelial tumours	128	77.57
2	Germ cell tumours	27	16.36
3	Sex cord stromal tumours	7	4.24
4	Miscellaneous	3	1.8
	Total	165	100

Table 10 shows among 165 neoplasms, surface epithelial tumours were the commonest neoplasm, comprised of 77.57% of the total neoplasms. Germ cell tumours were seen in 27 cases (16.36%). Sex cord stromal tumours were seen only 4.24% of total neoplasms. (Chart 4)

DISTRIBUTION OF SURFACE EPITHELIAL TUMOURS:

TABLE 11 Distribution of surface epithelial tumours

Serous tumours No of cases(%)			**Mucinous tumours No of cases (%)**		
Benign	**Borderline**	**Malignant**	**Benign**	**Borderline**	**Malignant**
65(76.47)	2(2.35)	18(21.17)	34(85)	0	6(15)
Total-85(100)			Total-40(100)		

As shown in Table 11.Serous tumours were more common (66.4%) among surface epithelial tumours, followed by Mucinous tumours(31.25%).Each case of Endometrioid carcinoma, Clear cell carcinoma, Brenner tumour(0.78%) was reported. (Chart 5)

Chart 4 Distribution of ovarian neoplasms based on histological types

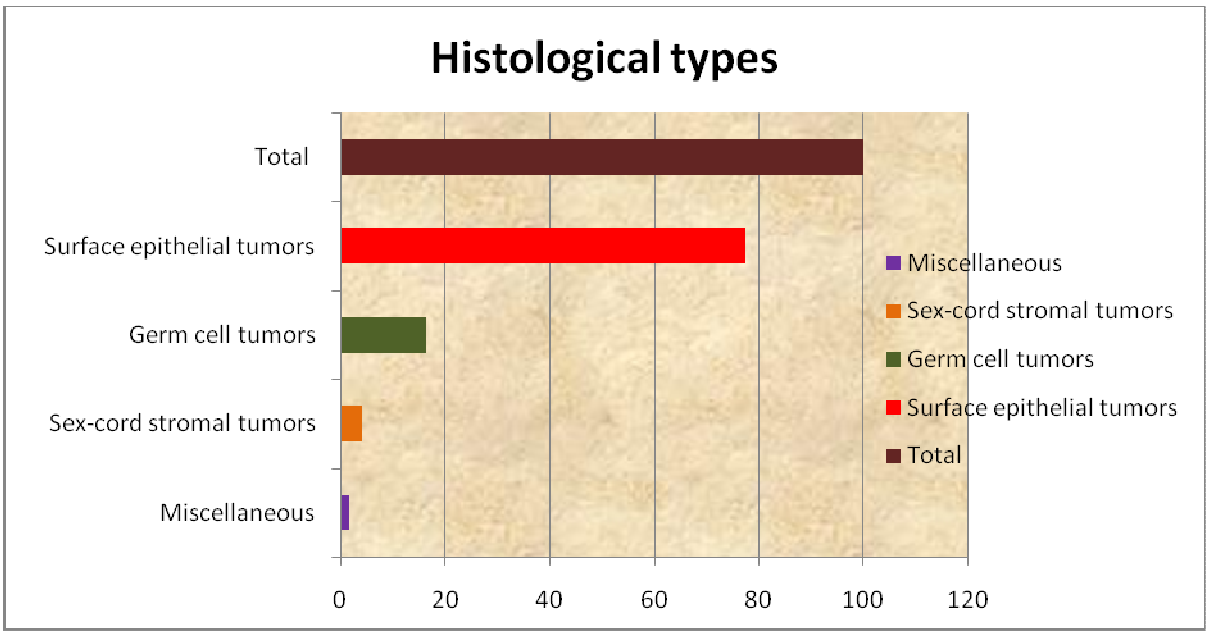
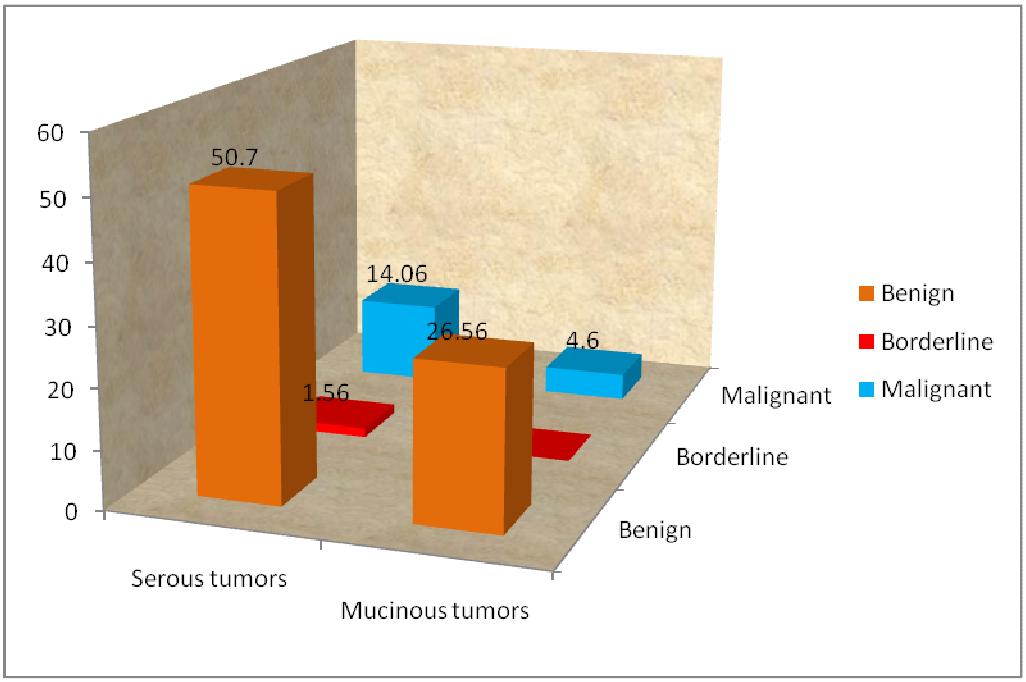


Chart 5 Distribution of surface epithelial tumours



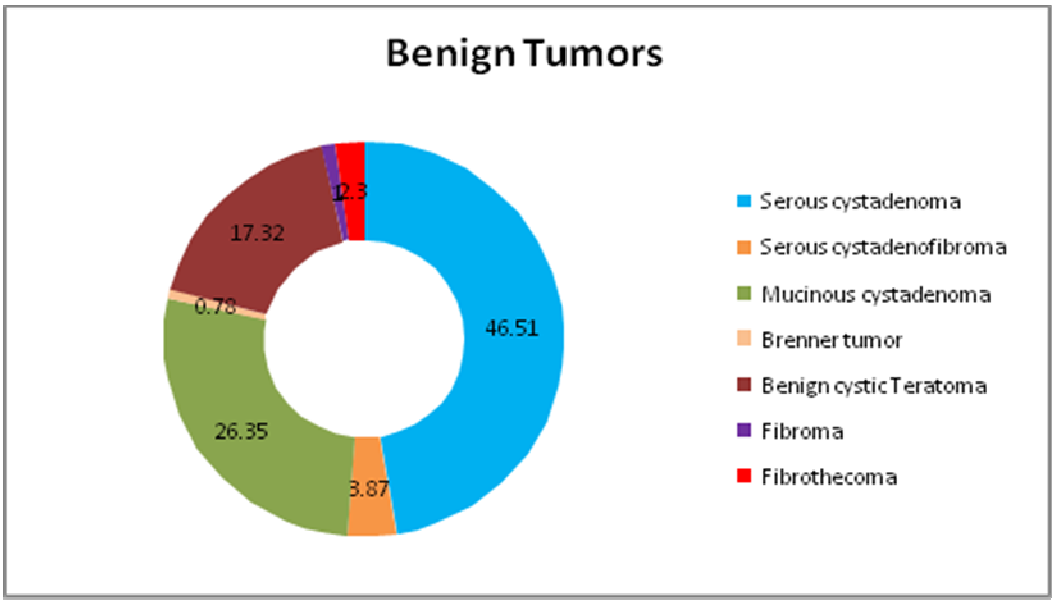
DISTRIBUTION OF BENIGN OVARIAN NEOPLASMS:

TABLE 12 Distribution of benign ovarian neoplasms

S.No	Types	No of cases	%
1	Serous cystadenoma	60	46.51
2	Serous cystadenofibroma	5	3.87
3	Mucinous cystadenoma	34	26.35
4	Brenner tumour	1	0.78
5	Dermoid cyst	22	17.32
6	Fibroma	2	1.57
7	Fibrothecoma	3	2.36
	Total	129	100

Present study showed that Serous cystadenoma was the commonest benign ovarian tumour seen in 60 cases (46.51%).Second most common tumour was mucinous cystadenoma (34cases,26.35%), followed by dermoid cyst (22 cases, 17.32%) (Table12)(Chart 6)

Chart 6 Distribution of benign ovarian neoplasms



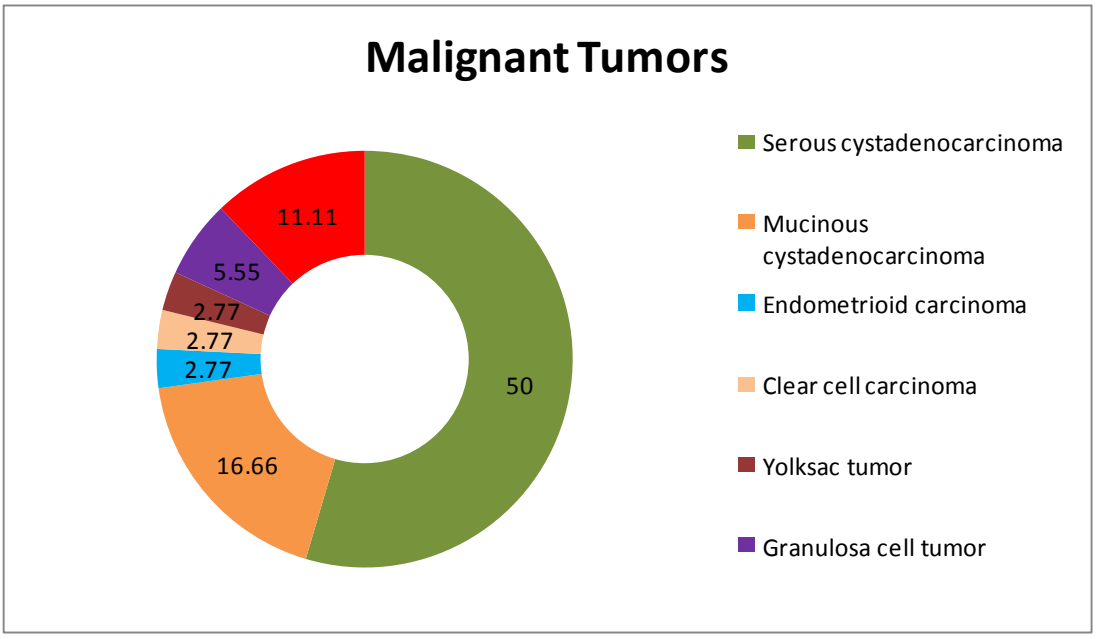
DISTRIBUTION OF MALIGNANT OVARIAN TUMORS

TABLE 13 Distribution of Malignant ovarian tumours

S.No	Histological types	No of cases	Percentage (%)
1	Serous cystadenocarcinoma	18	50
2	Mucinous cystadenocarcinoma	6	16.66
3	Endometrioid carcinoma	1	2.77
4	Clear cell adeno carcinoma	1	2.77
5	Yolk sac tumour	1	2.77
6	Malignant mixed germ cell tumour	4	11.11
7	Granulosa cell tumour	2	5.55
8	Miscellaneous	3	8.33
	Total	36	100

As shown in Table 13, serous cystadenocarcinomas were the commonest malignant ovarian tumour (18 cases, 50%).Mucinous cystadenocarcinomas was the second most common tumour (6 cases, 16.66%). (Chart 7)

Chart 7 Distribution of malignant ovarian tumours



DISTRIBUTION OF OVARIAN NEOPLASMS AMONG DIFFERENT AGE GROUPS

Table 14 Distribution of ovarian neoplasms among different age groups

S.No	Age group(years)	Surface epithelial tumours		Germ cell tumours		Sex cord-stromal tumours	
		No of cases	%	No of cases	%	No of cases	%
1	<19	0	0	5	18.51	0	0
2	20-39	48	37.5	13	48.14	3	42.85
3	40-59	57	44.53	8	29.62	4	57.14
4	>60	23	17.96	1	3.7	0	0
	Total	128	100	27	100	7	100

Surface epithelial tumours were common in the age group between 40-59years.Germ cell tumours were common in younger age group (10-40years) (Table 14)

DISTRIBUTION OF NON NEOPLASTIC LESIONS

TABLE 14 Distribution of non-neoplastic lesions

S.No	Types	No of cases	%
1	Follicular cyst	8	21.05
2	Luteal cyst	4	10.52
3	Inclusion cyst	3	7.89
4	Endometriotic cyst	20	52.63
5	Massive edema of ovary	1	2.63
6	Tuboovarian abscess	2	5.26
	Total	38	100

As shown in Table: 15, the most common non neoplastic cyst diagnosed was endometriotic cysts (20 cases, 52.63%), followed by follicular cysts (8 cases, 21.05%).

AGE OF THE PATIENTS AND SIZE OF THE NON NEOPLASTIC LESIONS:

TABLE 15 Age of the patient and size of the non-neoplastic lesions

S.No	Type of cysts	Age of the patient		Size of the lesion	
		Range	Mean	Range	Mean
		Years	Years	Cms	Cms
1	Endometriotic cyst	23-70	33.6	4-12	8
2	Follicular cyst	30-48	41.25	4-7	5.5
3	Corpus luteal cyst	27-38	33.25	4-12	8
4	Massive edema of ovary	48	48	5	5
5	Tuboovarian abscess	30-38	34	5-7	6
6	Inclusion cyst	22-77	47	6-7	6.5

In the present study the mean age of the endometriotic cyst was 33.6years, of follicular cyst was 41.22years, of luteal cyst was 33.25years, of inclusion cyst was 47years, of tuboovarian abscess was 47years.The mean diameter of endometriotic cyst was 8cms, of follicular cyst 5.5cms, of luteal cyst 8cms, of inclusion cyst was 6.5cms, of tuboovarian abscess was 6cms in diameter. (Table 16)

HISTOMORPHOLOGICAL PATTERNS OF OVARIAN LESIONS:

The details of the histomorphological types of benign ovarian tumours found in this study are given below.

SEROUS CYSTADENOMA:

This was the most common benign tumour found in this study. Totally, 60 cases were reported. Serous cystadenoma accounting for 36.36% of total ovarian neoplasms. Peak incidence was seen in 3rd and 4th decade of life. The youngest patient was 20years old and oldest was 68years old. All were unilateral tumours.

Macroscopically, the largest specimen was measured about 18x14x6cms and smallest was measured 5x2x2cms with a smooth external surface. Cut surface of the many tumours showed uniloculated cyst with smooth inner surface, containing serous fluid. Some tumours showed papillary excrescence from the inner surface.

Microscopically these tumours showed cyst wall, lined by cuboidal epithelium, few tumours showed simple papillae lined by cuboidal cells. In a few cases, cysts were lined with ciliated cuboidal cells.

SEROUS CYSTADENOFIBROMA:

In this study, only 5 cases were reported as serous cystadenofibroma. Largest tumour was measured about 12x8x4cms and smallest was measured about 5x3x2cms. All of them were unilateral tumours.

Macroscopically they showed solid and cystic cut surface. Solid areas are greyish white in colour, firm in consistency and cystic areas showed papillary projections from its inner surface. On microscopical examination, they showed a cyst wall thrown into papillae lined by cuboidal epithelium with subepithelial abundant fibrous tissue.

SEROUS BORDERLINE TUMOUR:

Only 2 cases were reported as borderline type, seen in 3rd and 4th decade of life. Macroscopically, they were unilateral, cystic masses. Cut section showed solid and cystic areas, with inner surface lined by papillary excrescence. Microscopical examination showed a cyst wall lined with branching papillary fronds lined by stratified cuboidal cells with mild nuclear atypia with minimal stromal invasion.

MUCINOUS CYSTADENOMA:

Present study showed 34 cases of mucinous cystadenoma (26.77% of all benign tumours).All the them were unilateral tumours. Most cases occurred in 4th and 5th decade, presented with abdominal mass. The youngest patient was 25years old and oldest was 70years old.

Macroscopically, they are cystic tumours; largest tumour was measured about 35x30x10cms and smallest measured 5x5x2cms with smooth outer surface. Cut surface showed multiloculated cysts containing mucoid substance with smooth inner surface. Microscopical examination showed a cyst wall lined by tall columnar epithelium resembling endocervical lining. Intracellular mucin was demonstrated with special stains.

BRENNER TUMOUR:

One case of Brenner tumour was reported in a 67 years postmenopausal woman, who was presenting with abdominal pain. Macroscopically it was unilateral and measured about 11x10x7cms. Cut surface showed solid tumour, greyish white in colour, firm in consistency.

Microscopically, the tumour cells are arranged in nests surrounded by dense fibroblastic stroma. The tumour cell nuclei showed longitudinal grooves. (Figure13)

FIBROTHECOMA:

In the present study we noted 3 cases of Fibrothecoma and two cases of Fibroma. All the cases were seen in 4^{th} and 5^{th} decade. Fibromas were small in size measured about 5x4x2cms. But the mean size of fibrothecomas was 12cm in diameter. All were unilateral tumours. Macroscopically, they were well circumscribed solid tumours, greyish white to yellow in colour, firm in consistency.

On microscopical examination, fibromas were arranged in fascicles of spindle shaped cells with bland nuclei admixed with bundles of collagen. Fibrothecoma showed the tumour cells were plump oval to spindle shaped cells with eosinophilic cytoplasm containing bland nuclei. The slide was stained with Reticulin stain, reticulin fibres were demonstrated around the individual cells. (Figure 18, 19)

BENIGN CYSTIC TERATOMA:

In our study it contributes 13.33% of total ovarian neoplasms and 81.48% among germ cell tumours. Mean age at presentation was 34.5years, youngest patient was 15years old and oldest patient was 70years woman. All the tumours were unilateral except two cases, which are bilateral.

Macroscopically the smallest tumour measured about 5x2x2cms, largest tumour measured about 5x2x2cms. All the cases were cystic in consistency; few cases were predominantly cystic and solid in consistency. In majority of the cases cut surface showed sebum, hair and teeth. Microscopically it showed mature elements of all the three germ cell layers.

HISTOPATHOLOGICAL PATTERN ANALYSIS OF MALIGNANT TUMOURS:

Table 16 Microscopic patterns of malignant tumours

Type of cancers	Microscopical patterns	No of cases
Serous cystadenocarcinoma	Complex branching papillary pattern	11
	Solid sheets	7
Mucinous cystadenocarcinoma	Mixed complex papillary and glandular pattern	6
Endometrioid carcinoma	Glandular pattern	1
Clear cell carcinoma	Solid sheets	1
Granulosa cell tumour	Micro and macrofollicular pattern	1
	Solid sheets	1
Yolk sac tumour	Microcystic and reticular pattern	1
Metastatic tumour	Nodular growth composed of glands	1
Struma carcinoid	Trabecular pattern	1

IMMUNOHISTOCHEMICAL FINDINGS:

Table17 Immunohistochemical findings of malignant tumours

Type of tumours	Immuhistochemistry	No of cases
Poorly differentiated serous carcinomas	WT-1 positive	7
	CK 7 positive	5
Bilateral Mucinous cystadenocarcinoma	CK 7 positive	4
	CK 20 negative	4
Endometrioid carcinoma	CK 7 and EMA positive	1
Granulosa cell tumour	Inhibin positive	1
Yolk sac tumour	AFP positive	1
Dysgerminoma	PLAP	1
Metastasis	CK 7 negative	1
	CK 20 positive	1

SEROUS CYSTADENOCARCINOMA:

Out of 36 ovarian malignancies, 18 cases were reported as serous cystadenocarcinoma. Peak incidence of age was 5^{th} and 6^{th} decade of life. Most of them were bilateral tumours.

Macroscopically, the largest specimen was measured about 18x15x10cms and smallest measured as 6x6x6cms. In majority of the cases, we received irregular solid masses. Cut surface showed variegated in appearance with areas of haemorrhage and necrosis.

Microscopic examination showed that the tumour composed of cells arranged in complex papillary pattern, papillae were lined by pleomorphic cells with atypical

nuclei infiltrating into the underlying stroma, admixed with areas of haemorrhage and necrosis.

Out of 18 cases, 7(38.8%) cases were poorly differentiated type. All of them were presented in postmenopausal age group (more than 60years). Macroscopically, they were all bilateral, solid tumours with capsular breach and omental deposits were seen in all the cases. Microscopically, they show solid sheets of small hyper chromatic cells with numerous mitotic figures, few cases show syncytial aggregates of tumour gaint cells. All the slides of poorly differentiated type were subjected with immunomarker CK 7 and WT-1.WT-1 expressed nuclear positivity in all the cases, whereas CK 7 was positive in five cases.(Table 16,17) (Figure 4,5,6)

MUCINOUS CYSTADENOCARCINOMA:

Four cases of mucinous cystadenocarcinoma were reported in this study. It was seen in 5^{th} and 6^{th} decade. Out of 4 cases, 3 of them showed bilateral presentation. Macroscopically they were all large tumours measured about 30x20x10cms with bosselated surface. Cut surface showed closely packed multilocular cystic spaces filled with mucinous fluid admixed with greyish white solid areas showing haemorrhage and necrosis.

Microscopically, they showed a tumour arranged in complex papillary pattern and irregular closely packed glands lined by stratified pleomorphic cells with atypical nuclei admixed with extracellular pool of mucin. Intracellular and extracellular mucin was demonstrated by special stains-Periodic acid Schiff stain. It showed magenta colour. All the bilateral tumours were stained by Cytokeratin 7 and CK 20 to rule out the gastrointestinal primary. Tumour cells exhibit CK 7 positivity, confirmed the ovarian origin. (Table 16, 17) (Figure7,8,9,)

ENDOMETRIOID CARCINOMA:

One case of 52 year female presented with abdominal mass and pain was reported as endometrioid adenocarcinoma. Macroscopically, the tumour measured about 10x9x7cms. Cut surface showed predominantly solid areas, which is greyish white in colour admixed with areas of haemorrhage and necrosis.

Microscopic examination it showed, irregular glands, lined by stratified columnar cells with marked pleomorphism and atypical nuclei. Micro glandular structures resemble that of granulosa cell tumour. Immunostainig with CK 7 and EMA was performed, to rule out granulosa cell tumour and they were found to be positive. (Table 16, 17)(Figure 10, 11)

CLEAR CELL CARCINOMA:

Only one case of clear cell carcinoma was reported in this study. It was seen in the 55years old postmenopausal women presented with abdominal pain with mass. Tumour measured about 20x10x8cms.It was unilateral. Macroscopically, it was a solid tumour, spongy in consistency admixed with massive areas of haemorrhage and necrosis. Microscopic examination it showed that sheets of polygonal cells with clear cytoplasm containing pleomorphic nuclei. Slide was stained with PAS, cytoplasm showed magenta colour. (Table 16)(Figure 12)

GRANULOSA CELL TUMOUR:

In our study we found, two cases of Granulosa cell tumours. They were seen in younger age group with the mean age of 30years.One patient was presented with bleeding per vagina with abdominal pain. Both were unilateral tumours. One tumour was small in size, measured 7x6x6cms whereas another one was measured about 22x19x10cms.

On microscopical examination, one tumour was arranged in both micro and macro follicular pattern, cells were having bland nuclei with intra nuclear grooves. Another tumour showed solid and trabecular pattern of growth, the tumour cells revealed nuclear grooves. Mitotic activity was less in both the tumours. Reticulin stain was performed in this case and reticulin fibres were demonstrated around the each tumour cell clusters. Immustaining with Inhibin expressed nuclear positivity (Table 16, 17) (Figure 14,15,16,17)

MALIGNANT MIXED GERM CELL TUMOUR:

Four cases were reported in our study, contributed 2.42% of all ovarian neoplasms. Three cases were seen in younger age group with mean age of 19years and one case was 47 years old woman. All the cases were unilateral tumours. Mean size of the tumour was 15 cms in diameters. Tumours of younger age group presented with a large mass.

Macroscopically, all the tumours showed solid or solid-cystic consistency. Microscopically three tumours were showing Yolk sac tumour with Dysgerminoma. One case showed the mixture of Immature teratoma and Embryonal carcinoma. Yolk sac tumour showed reticular and micro cystic pattern of growth admixed with Schiller Dual bodies. Immunostaining with AFP showed positivity. (Table 16, 17) (Figure 20,21,22,23)

METASTATIC TUMOURS:

Two cases of metastatic tumours were reported in this study. These are presented in 4^{th} and 6^{th} decade. Both the tumours were bilateral. The mean size of the tumour was 8cms in diameter. Macroscopically one tumour was asymmetrically enlarged, cut surface showed greyish white in colour, solid in consistency. Another tumour had solid-cystic cut surface.

Microscopically both the tumours showed nodular pattern of growth composed of cells arranged in infiltrative, glandular pattern admixed with dirty necrosis. In addition to this, one tumour showed cordlike arrangement of the signet ring cells. This tumour designated as Krukenberg tumour and it shows PAS positivity. The tumour with glandular pattern of growth was subjected with Cytokeratin 20 &7. It showed CK 20 positivity, confirms the gastrointestinal origin. (Table16, 17) (Figure 24,25,26,27)

One case of Struma carcinoid was seen 52 year postmenopausal women. Unilateral tumour with the size of 10cms in diameter. Cut surface showed multiloculated cyst containing brownish glistening solid areas. Microscopical examination showed tumour arranged in trabecular pattern with a focus showing insular pattern composed of small hyperchromatic cells intermixed with thyroid follicles, showing intact capsule.

NON NEOPLASTIC LESIONS:

Endometriotic cyst:

This was the most common non neoplastic cyst found in this study (52.63%). Most commonly it was seen in 2nd and 3rd decade and abdominal pain was the common presentation. All were unilateral cysts with a mean size of 8cms in diameter. Macroscopically, most of the cysts were uniloculated with haemorrhagic content. Microscopical examination showed a cyst wall lined by attenuated endometrial glands admixed with stroma showing hemosiderophages and haemorrhages.

Follicular cyst:

This was the second most common lesion, contributes 21% of total non neoplastic lesions. The mean age of presentation was 41years.Majority of the cases were unilateral. The mean size of the tumour was 5.5cms in diameter. Three patients

were presented with abnormal uterine bleeding. Microscopically, cysts were lined by inner granulosa cells and outer layer of theca cells.

Corpus Luteal cyst:

It accounts for 10.52% of non-neoplastic lesions. The mean age of presentation was 30years.The mean size of the cyst was 8cms in diameter. Macroscopically, the cyst wall is convoluted and yellow in colour. Microscopically, it was lined by luteinised granulosa cells interrupted by wedges of theca lutein cells.

One case of massive edema of ovary was reported. Cut section showed homogenous, glistening appearance. Microscopically, edematous ovarian stroma was seen, sparing the cortex. (Figure 28, 29).

DISCUSSION

INCIDENCE OF OVARIAN CANCERS:

Incidence of ovarian cancers is more common in Western countries than Asian countries. A study conducted by Koonings et al[1989][3], in United States showed that the frequency of ovarian cancers among all gynaecological cancers was 15.8%,whereas another study done in Nepal by Kayastha et al[2009][38] reported 9.5% of incidence among 568 cases and Santhosh Kumar Mondal et al[2011][39]reported 8.7% in Eastern India. When compared to western data, our study showed a lesser incidence [8.2%], but correlates well with Asian statistics.

DISTRIBUTION OF NEOPLASTIC AND NON NEOPLASTIC LESIONS:

A study conducted by De Kroon et al [2004][40] in United States reported that two-third of ovarian cysts were found to be non-neoplastic. In India [Meerut], Gupta et al [2007][41] reported 58.79% of non-neoplastic lesions and 41.2% of neoplastic lesions among 282 cases, whereas another study done in Mumbai by Bhattacharya et al [1980][42]reported that out of 270 cases, 7.41% were non neoplastic, 92.59% were neoplastic. The present study showed that, out of 203 cases, 38(18.71%) cases were non neoplastic, 165(81.28%) cases were neoplastic lesions. Our study correlates with the study of Bhattacharya et al, not with other studies.

DISTRIBUTION OF BENIGN, BORDERLINE AND MALIGNANT NEOPLASMS:

Major fraction of ovarian tumours in our study comprises benign tumours (76.96%) followed by malignant (21.81%) and borderline tumours (1.21%).Comparison of percentage of our results with various studies shown in Table18.

Table 18 Comparison of percentage of Benign, Borderline and Malignant tumours with other studies:

S.No	Authors	Benign tumours	Borderline tumours	Malignant tumours
1	Bhattacharya et al[1980][42]	64.07%	7.41%	28.52%
2	Koonings et al[1989][3]	75.45%	3.6%	20.9%
3	Santhosh Kumar Mondal et al [2011][39]	63.1%	7.3%	22.9%
4	Gupta N et al[2007][41]	72.9%	4.1%	22.9%
5	Pilli et al [2005][43]	76%	2.8%	21.2%
6	Present study	76.96%	1.21%	21.81%

Results of our study were similar to the findings of other studies (Table 19) but higher number of borderline cases has been reported by Santhosh Kumar Mondal et al[39]and Bhattacharya et al[42].

DISTRIBUTION OF OVARIAN TUMORS ACCORDING TO AGE:

In the United States, Koonings et al [US] [1989] reported that 94% of benign tumours occurred between 20 – 29 years and 92% of malignant tumours occurred after sixth decade. In our study, 44.09% of benign tumours were diagnosed between 20-40 years of age, whereas 77.77% malignant tumours occurred after fourth decade. Present study correlates with the studies conducted by Santhosh Kumar Mondal et al [Eastern India] [2011][39,] Kayastha et al [Nepal] [2009][38], Bhattacharya et al [Mumbai] [1980][42] When compared to Western countries, our study shows earlier age of onset of malignant tumours.

In our study we noted that most of the epithelial tumours were seen between 4th and 5th decade. Our result was comparable with the studies conducted in India by Santhosh Kumar Mondal et al[2011] [39]and Bhattacharya et al[1980][42].But the studies carried out in Western population by Koonings et al[US][1989][3] and Di Bonito et al[Italy] [1988][44] found that epithelial tumours were more common after 50 years. Cannistra et al[England][2004][9]reported that 58% of the germ cell tumours occurred before 30 years and Santhosh Kumar Mondal et al[Eastern India][2011][39] reported 67.77%.These observations correlate with our study where 66.65% of the germ cell tumours occurred in the age group of less than 30years.

MODE OF PRESENTATION:

In the present study we noted that almost half of the cases (47.27 %) presented with combined abdominal pain and mass and 37.17% of the cases presented only with abdominal pain. Comparative analysis was done with various studies and it was shown in Table 19

Table 19 Mode of presentation of cases of various studies in comparison with our study

S.No	Mode of presentation	Authors			
		Maheswari et al	Couto et al	Pilli et al	Present study
1	Abdominal pain	39%	40%	70%	31.51%
2	Abdominal mass	71%	90%	63%	13.93%
3	Bleeding per vagina	3%	39%	10%	3.03%
4	Ascites	3%	4.9%	24%	0%

Our study correlated well with the studies done by Pilli et al[Belgaum][2005] and Kayastha et al [Nepal][2009][38], where abdominal pain was the commonest symptom, and cases presenting with abdominal mass were less in the present study compared to the studies carried out by Maheswari et al[Aligarh][1994][16] and Couto et al[Goa][1993][45].Studies conducted in Western world by Bankhead et al[UK][2008][46] and Goff et al[US][2004][47] showed that abdominal distension was the commonest symptom.

LATERALITY:

Present study showed that 89.7% were unilateral tumours and 10.3% were bilateral, thus correlating well with other studies conducted by Couto et al[Goa][1993][45] and Prabhakar et al[Amristar][1989][1] whereas Koonings et al[US][3] and Kar et al[Cuttack][2005][48] found more number of bilateral tumours 22% and 26.8% respectively.

Table 20 Comparison of laterality of neoplastic lesions with other studies

Sino	Authors	Benign(%)		Borderline(%)		Malignant(%)	
		U/L	B/L	U/L	B/L	U/L	B/L
1	Jha et al [2008][49]	93.33	6.67	0	0	57.7	42.3
2	Kuladeepa et al[2011][50]	93.75	6.25	80	20	68.42	31.58
3	Kayastha et al[2009][38]	93.02	6.97	100	0	33.33	66.66
4	Present study	97.63	2.36	100	0	61.11	38.88

As shown in Table 20 present study showed that majority of benign tumours (97.63%) were unilateral and most of malignant tumours were bilateral(38.88%). This observation is in concurrence with the above mentioned studies.

GROSS FINDINGS:

In our study, we noted that out of 165 neoplastic cases, 66.99% of the tumours were cystic, 10.3% of the tumours were solid and 22.66% of the tumours had both solid and cystic cut surface. All the benign tumours were cystic and all the malignant tumours showed either solid or solid-cystic cut surface. This observation was correlated with the studies by Koonings etal [US] [1989][3], Gupta et al [Meerut] [2007][41], Couto et al [Goa] [1993][45].

DISTRIBUTION OF OVARIAN TUMORS ON THE BASIS OF MICROSCOPICAL DIAGNOSIS:

In our study we found that Surface epithelial tumours comprised 77.57% of all ovarian neoplasms, Germ cell tumours contribute 16.36%, Sexcord stromal tumours contribute 4.24% and metastatic tumours were 1.21%.This result correlates with the studies done in Western population by Koonings et al[US][1989][3],Di Bonito et al [Italy][1988][44],in Asian population by Samina Zaman et al[Pakistan][51],Kayastha et al[Nepal][38],Pilli et al[India][43].But in contrast Gupta et al[Meerut][2007][41] found lower incidence of epithelial tumours and a higher incidence of germ cell and metastatic tumours .

Koonings et al[3] stated that epithelial tumours contribute 40% of the all benign neoplasms and 85% of malignant neoplasms. Di Bonito et al[44] also reported that 50% of the benign tumours and 94.4% of the malignant tumours were of epithelial origin. This is in contrast to our study in which epithelial tumours comprised 78.74% of total benign neoplasms and 72.22% of malignant neoplasms. This observation was comparable with the studies conducted by Samina Zaman et al[51], Swamy et al, Pilli et al[43].

Present study showed that germ cell tumours contribute 17.32% of benign neoplasms and 13.88% of malignant neoplasms. This result was similar to the studies done in Indian population by Couto et al[45] and Prabhaker et al[1]. But Koonings et al[3] reported more number of benign (45%) and less number of malignant germ cell tumours (4.3%).Di Bonito et al[44] also found that 27.5% of benign and 2.8% of malignant germ cell tumours. This observation was not in concurrence with our study.

SEROUS TUMOURS:

DISTRIBUTION OF SEROUS TUMORS WITH RESPECT TO ALL OVARIAN TUMORS

Studies conducted by Couto et al[45] ,Kayastha et al[38] and Pilli et al[43]observed 42.5%, 40% and 42.9% of serous tumours among total ovarian neoplasms respectively. This observation correlates with our study (39.39%), but in contrast to our study, Misra et al [UP] [1991][52](55.8%) reported higher incidence of serous tumours .The present study showed, out of 85 cases 58 cases (69.4%) were serous cystadenomas, 5 cases were serous cystadenofibromas, one case was papillary serous cystadenoma, 2 cases were borderline tumours and remaining 18 cases were carcinomas. Saxena HMK et al [1980][53]and Maheswari et al [1994][16]demonstrated 72.03% and 69.8% of serous cystadenomas in their studies respectively analogous to our study. Microscopically benign tumours showed a cyst wall lined by cuboidal epithelium with a few cases (5 cases) showed ciliated cells. In contrast, Krigman et al [US] [1994][54] observed more ciliated cells.

In our study Serous cystadenocarcinoma constitutes 21.17% of all serous tumours which was comparable to the study of Maheswari et al (19.8%).In contrast to our study, Krigman et al[54] reported 36% of malignant serous tumours .All the cases either showed solid or solid- cystic cut surface with areas of haemorrhage and

necrosis as observed by Krigman et al[54].Out of 18 cases 7 cases were poorly differentiated type and majority of these cases were postmenopausal women(above 60 years of age) with bilateral presentation. Dehari et al [US] [2007][55] reported that two third of poorly differentiated tumours were bilateral which correlates with our study. All the seven cases showed nuclear positivity with WT-1 marker, 5cases expressed membrane positivity with CK-7.This observation was similar to the study done by Hussaini et al[US][2004][56].It was a immunohistochemical study done among 76 cases, all the 38 cases of ovarian serous carcinomas, showed WT-1 nuclear positivity.

Gupta et al [2007][41] reported that borderline tumours contribute 1% of total ovarian neoplasm which was comparable with our study (1.2%). But Pilli et al [2002][44] and Santhosh Kumar Mondal et al [2011][39] reported higher incidence of borderline tumors. Microscopically they showed epithelial stratification, nuclear atypia and minimal stromal invasion. McKenney et al [2006][57] found that presence of micro invasion was associated with poor prognosis.

MUCINOUS TUMOURS:

COMPARISON OF MUCINOUS TUMOURS WITH RESPECT TO ALL OVARIAN TUMORS:

The present study showed that mucinous tumours accounting for 24.24% of all ovarian neoplasm. This observation was in accordance with the studies by Couto et al[1993][45],Gupta et al[2007][41]but Misra et al[1991][52] reported less number of mucinous tumours in comparison with our study. Out of 40 mucinous tumours 36 (90%) were benign, 4 (10%) cases were malignant. There were no borderline tumours reported. But Koonings et al[1989][3]demonstrated less number of (74.5%)benign tumors,10% of borderline tumours and more number of (15%) malignant tumours as compared with our study. Macroscopically, size of the tumours varied from 5cms in

diameter to 35 cms in diameter. Tyagi SP et al [1993][58] reported a case with a tumour of 47 cm in diameter, and showed 10 % bilateralism among mucinous tumors. In the present study we noted 6.4 % bilateralism. All these tumours were malignant. In our study 89.8% of the tumours were multilocular this is comparable with the study done by Maheswari et al [1994][16](83.9 %). Microscopically, benign tumours showed a cyst wall lined by tall columnar cells with apical mucin which was demonstrated by special stain. Krigman et al [US] [1994][54] observed all the malignant tumours and 10% of the benign tumours were of intestinal type. However in the present study all the benign tumours were lined by endocervical type epithelium and malignant tumours were lined by intestinal type epithelium. Vang et al [US] [2006][59] analysed, CK 7 and CK 20 expression among 179 mucinous tumours and found that primary mucinous carcinomas exhibit CK 7 positivity and C K 20 negativity. In our study all the bilateral mucinous carcinomas were subjected with CK 7 & CK 20 to rule out the intestinal metastatic deposits and found to be CK 7 positive and CK 20 negative.

OTHER SURFACE EPITHELIAL TUMOURS:

In our study endometrioid carcinoma contributes to 0.78 % of surface epithelial tumors.This incidence correlates with study conducted by Prabakar et al[1989][1].He demonstrated 1.1% of endometrioid carcinomas whereas studies by Maheswari et al[1994][16] and Dawar et al[2004][60] reported higher incidences(3.65%&5.7% respectively).Vigano et al[2008][18] demonstrated that association of endometriosis in 10% of the tumours , but no associated endometriosis was seen in our case Immunomarkers of CK 7 and EMA were positive in our study, similar to the study done by Riopel et al [1998][61].

In our study we reported one case of clear cell carcinoma. It contributes, 0.78% of surface epithelial tumours, which correlates with the study of Maheswari et al[16] (0.79%).

There was one case of benign Brenner tumour accounting for 0.78 % of surface epithelial tumours. But Krigman et al [1989][3]and Maheswari et al [1994][16] reported 2%, and 1.1% respectively. Microscopically the tumour cells were arranged in nests surrounded by fibrous stroma. The tumour cells resemble that of urothelial cells as stressed by Balasatw et al [1997][62].

GRANULOSA CELL TUMOR:

The present study showed that granulosa cell tumour contributes 1.21 % (2 cases) of all ovarian neoplasms. The incidence was less compared to the study done by Ramachandra et al [1972][63](2.7 %). Mean age of presentation in our study was 30.5 years which was less compared to the study done by Ayhan et al[2009][64] (47 years). Macroscopically, one tumour was measured 22 cms in diameter; another was measured 7 cms in diameter. Ayhan et al[64] stated that the tumours larger than 15 cm had poor prognosis. Microscopically one tumour showed solid pattern, another tumour showed combined micro follicular and macro follicular patterns. Both the tumours showed low mitotic index. Stuart et al [2003][21] stated that microscopical patterns do not predict the prognosis and he also demonstrated Inhibin positivity in all the cases. In the present study Inhibin immunostaining was performed and found to be positive.

BENIGN CYSTIC TERATOMA:

Our study showed that benign cystic teratoma contributes 13.33% of total ovarian neoplasm and 81.48% of total germ cell tumours .This observation correlates with the studies conducted in India by Couto et al[45],Tyagi et al[58] with an incidence of

15.45%, 18.46% of total ovarian tumours respectively. In a study conducted by Koonings et al[3], it contributed 95 % all germ cell tumours which was higher than our study. The present study showed 9 % tumours were bilateral which was similar to a study conducted by Tyagi et al[58] where 8.33% of tumours were bilateral. Majority of the cases in our study were reported in second and third decades of life as compared to the study done by Couto et al[45].

MALIGNANT MIXED GERM CELL TUMOR:

Malignant mixed germ cell tumours contributed about 2.4% of all ovarian neoplasms. Similar studies carried out by Prabakar et al[1] and Gupta et al[41] showed an incidence of 0.78% and 0.59% respectively which are less as compared with our study. We had 3 cases of yolk sac tumour with dysgerminoma and one case of immature teratoma with embryonal carcinoma. Kurman et al[1976][30]studied about 30 ovarian mixed Germ cell tumours and showed that Dysgerminoma was the most common constituent followed by Yolk sac tumour which correlates with our study. He also stated that if more than one third of a stage I neoplasm composed of Yolk sac tumour or Choriocarcinoma or Grade III Immature teratoma showed poor prognosis. In the present study we observed that more than one third of the tumour was composed of Yolk sac tumour in all the slides.

MISCELLANEOUS TUMOURS:

Metastatic tumours:

One case of Krukenberg tumour and another case of intestinal metastatic deposits were seen. It accounts for 1.21% of total neoplasm. But studies done by Couto et al[45], Mishra et al[52] and Prabakar et al[1] showed an incidence of 1.46 %, 1.07 % , 1.57 % respectively which were higher than our study. Krukenberg tumour was bilateral similar to a study done by Powari et al[2003][31].Microscopically diffuse

infiltration of tumour cells were seen, they arranged in nests or pseudo glandular pattern. In Krukenberg tumour, many signet ring cells were seen in oedematous ovarian stroma. The tumour cells were PAS positive. Holtz and Hart [1982][65]demonstrated that out of 27 cases 16 cases were PAS positive. Metastatic tumour showed Cytokeratin 20 positivity and Cytokeratin 7 negativity, confirming the gastrointestinal origin. This observation was similar to the study done by Vang et al [2006][66]and Baker et al [2005][67].Theyshowed that all the cases of metastatic tumours of intestinal origin were CK 20 positive and CK 7negative.

One case of Struma carcinoid was reported.Microscopical examination showed that in more than half of the tumour, the small cells were arranged in trabecular pattern admixed with thyroid follicles. Robby SJ et al [1980][68]analysed 50 cases and stated that the most common histological pattern present in Struma carcinoid was trabecular pattern.

NON NEOPLASTIC LESIONS:

INCIDENCE:

Table 21 Comparison of incidence of non neoplastic lesions with other studies.

S.No	Non neoplastic cysts	Authors		
		Samina Zaman et al[51]	**Pudasaini et al[69]**	**Present study**
1	Follicular cyst	22.24%	4.8%	3.9%
2	Corpus luteal cyst	34.83%	13.7%	1.9%
3	Endometriotic cyst	10.78%	5.9%	9.8%

In the present study endometriotic cyst was the most common non-neoplastic lesion, contributes 9.8% of total ovarian lesions(52.63% of non-neoplastic lesions).

This result contradicts other studies done by Samina Zaman et al[2010] [51]and Gupta et al[2007][41]where almost half of the cases were contributed by Luteal cyst.(Table 21)

MEAN AGE OF NON NEOPLASTIC LESIONS

In the present study we found that they were commonly occurred between 3rd and 4th decade. This observation correlates with the studies conducted by Samina Zamina et al [2010][51]and Pudasaini et al [2011][69]. (Table 22)

Table 22 Comparison of mean age of non-neoplastic lesions with other studies

S.No	Non neoplastic cysts	Authors		
		Mean age(years)		
		Samina Zaman et al	Pudasaini et al	Present study
1	Endometriotic cyst	30.28	35.28	33.6
2	Follicular cyst	37.67	39.1	41.25
3	Luteal cyst	35.41	32.12	33.25

GROSS FINDINGS:

Table23 Comparison of mean size of non neoplastic lesions with other studies

S.No	Non neoplastic cysts	Authors		
		Size (cms)		
		Samina Zaman et al[51]	Gupta et al[41]	Present study
1	Endometriotic cyst	5.98	5.5	8
2	Follicular cyst	3.95	5.2	5
3	Luteal cyst	3.76	6.1	8

As shown in Table 23 in our study showed that the mean size of the cysts was slightly larger than the above mentioned studies. In the present study abdominal pain was the commonest symptom, which was comparable with the studies by Gupta et al[41] and Wasim et al[70.]

SUMMARY AND CONCLUSION

A histopathological study of ovarian non neoplastic and neoplastic lesions was undertaken in the Department of Pathology Tirunelveli medical college to know the occurrence of different types of ovarian lesions in this region and found to be correlated with similar other studies conducted among Western and Asian population with only a few exceptions.

In the present study 203 ovarian lesions were analysed for the period of three years January 2008 to December 2010. Ovarian tumours were classified based on recent WHO classification.

Benign tumours were the major component of ovarian tumours constituting 76.96 % followed by malignant tumours (21.81 %) and borderline tumours (1.21%)

Surface epithelial tumours were the major group (77.57%), followed by germ cell tumours (16.36%) and sex-cord stromal tumours (4.4%).

These tumours occurred in all age group but most of the tumours were in the age group between 40-59years.Benign and borderline tumours occurred in younger age group whereas malignant tumours were common after 40years.The occurrence of malignancy is earlier than in Western statistics. Surface epithelial tumours occurred in all age groups. Germ cell tumours were common in younger age group.

Most of the tumours were unilateral 89.7%.and 10.3% of tumours were bilateral. Bilaterality was common among serous tumours. Most common presentation was abdominal pain.

The tumour varied in size, largest tumour was Mucinous cystadenoma measuring 35x30x18cms and smallest tumour was benign cystic teratoma measuring 5x2x2cms. Larger size was seen in benign mucinous tumours and malignant tumours.

All the benign tumours were cystic in consistency whereas, all malignant tumours had either solid or solid-cystic cut surface.

The commonest benign tumour was serous cystadenoma (47.24%) and the most common malignant tumour was serous cystadenocarcinoma (50%).

The occurrence of malignancy was more common in serous tumours 21.27% as compared to mucinous tumours 15%. Prevalence of poorly differentiated serous carcinoma was found to be high.

Germ cell tumours formed the second largest group (16.36%).Benign cystic teratoma was the commonest histological type. Malignant mixed Germ cell tumours contributed 1.81% of all ovarian neoplasms. One case was showing a rare combination of immature teratoma with embryonal carcinoma.

Two cases of granulosa cell tumours were reported. One case was presented with larger tumour size and solid pattern of growth with intact capsule.

Two cases of metastatic tumours were seen. One was Krukenberg tumour; another one was metastatic deposits from intestine.

All malignant tumours of our study showed capsular breach and areas of necrosis and haemorrhage except granulosa cell tumour and Krukenberg tumour.

Immunomarkers were applied in equivocal cases like all poorly differentiated serous carcinomas, bilateral mucinous carcinomas, endometrioid carcinoma, Germ cell tumours, and metastatic tumours to confirm the diagnosis. Ancillary studies – Special stains and Immunohistochemistry provide a valuable information for confirming the diagnosis.

Out of 203 ovarian lesions, 38 cases were non neoplastic lesions. Endometriotic cyst was most common comprising of 20cases (52.63%), followed by follicular cysts (21.05%).

Majority of the non-neoplastic lesions were unilateral cysts. Most commonly occurred in third decade and all the lesions were below 10cms.Most commonly presented with abdominal pain.

Ovaries are prone for occurrence of neoplasms which at the older age group, it gives enormous mental burden and economic drain for the individual. In the younger age group it may hinder the active life. Hence it is essential to differentiate neoplasms from non-neoplastic conditions and to know the prevalence of common ovarian neoplasms in a tertiary care set up. In our study both non neoplastic and neoplastic lesions were analysed based on age, clinical features, laterality, gross findings and histological studies. They correlated well with the studies undertaken in different parts of India both in frequency and prevalence.

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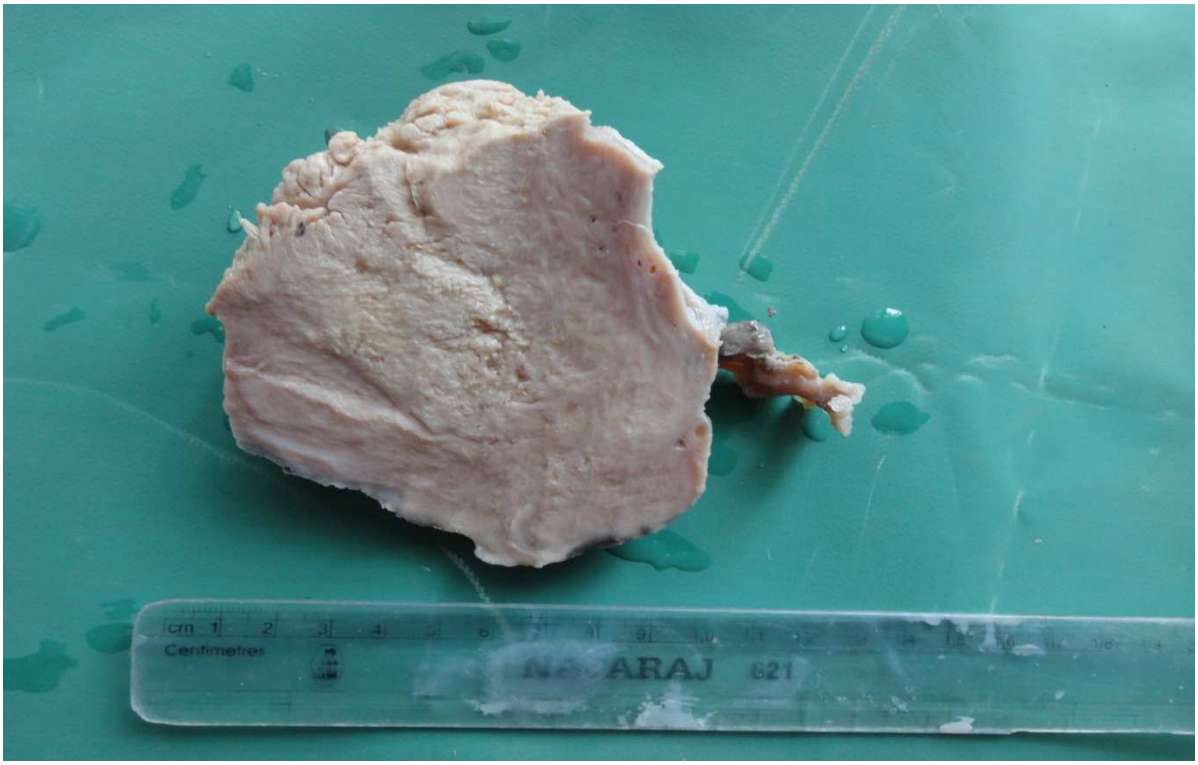

Figure 4 Poorly differentiated serous carcinoma-*Cut surface showing irregular, solid, greyish white mass with capsular breach.*

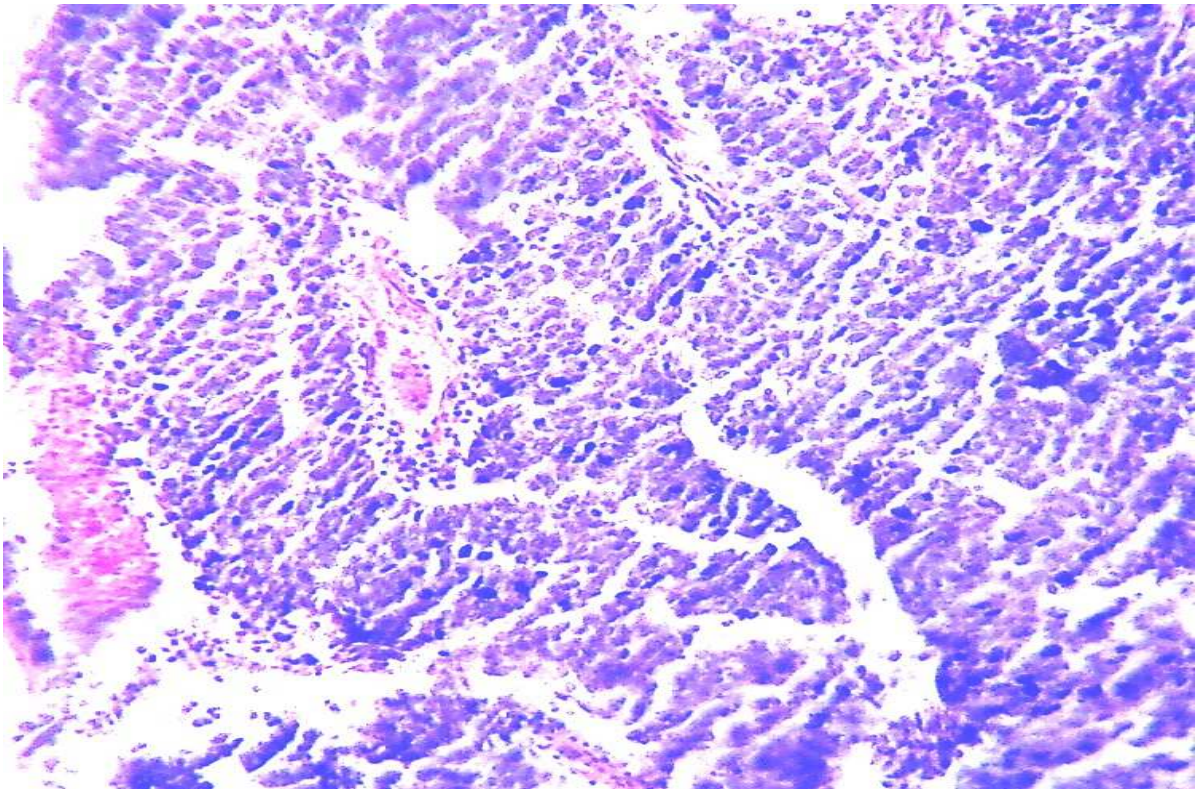

Figure 5 Poorly differentiated serous carcinoma-*Photomicrograph shows solid sheets of tumour cells containing irregular hyperchromatic nuclei admixed with necrosis. (H&E 100 X)*

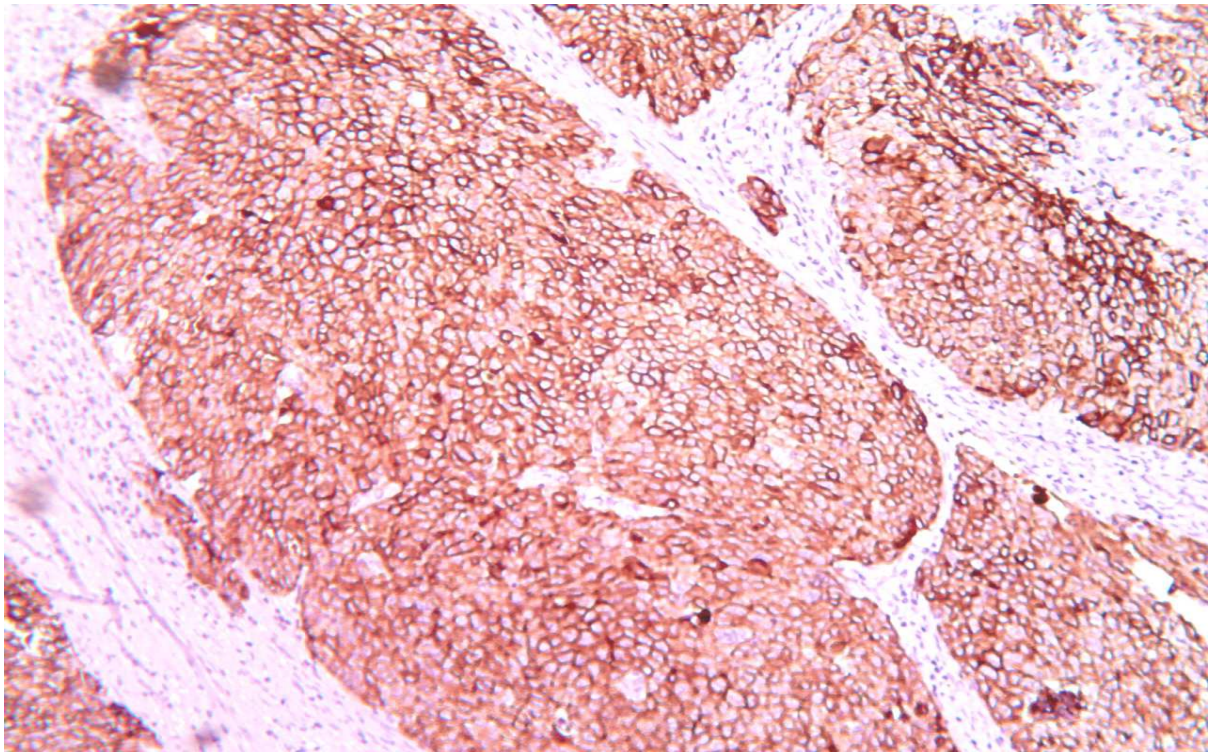

Figure 6 Poorly differentiated carcinoma- ***Photomicrograph showing strong membrane positivity of tumour cells for CK 7. (Immunostain CK 7 100 X)***

Figure 7 Mucinous cystadenocarcinoma- ***Gross picture showing multiloculated closely packed cysts containing mucoid material.***

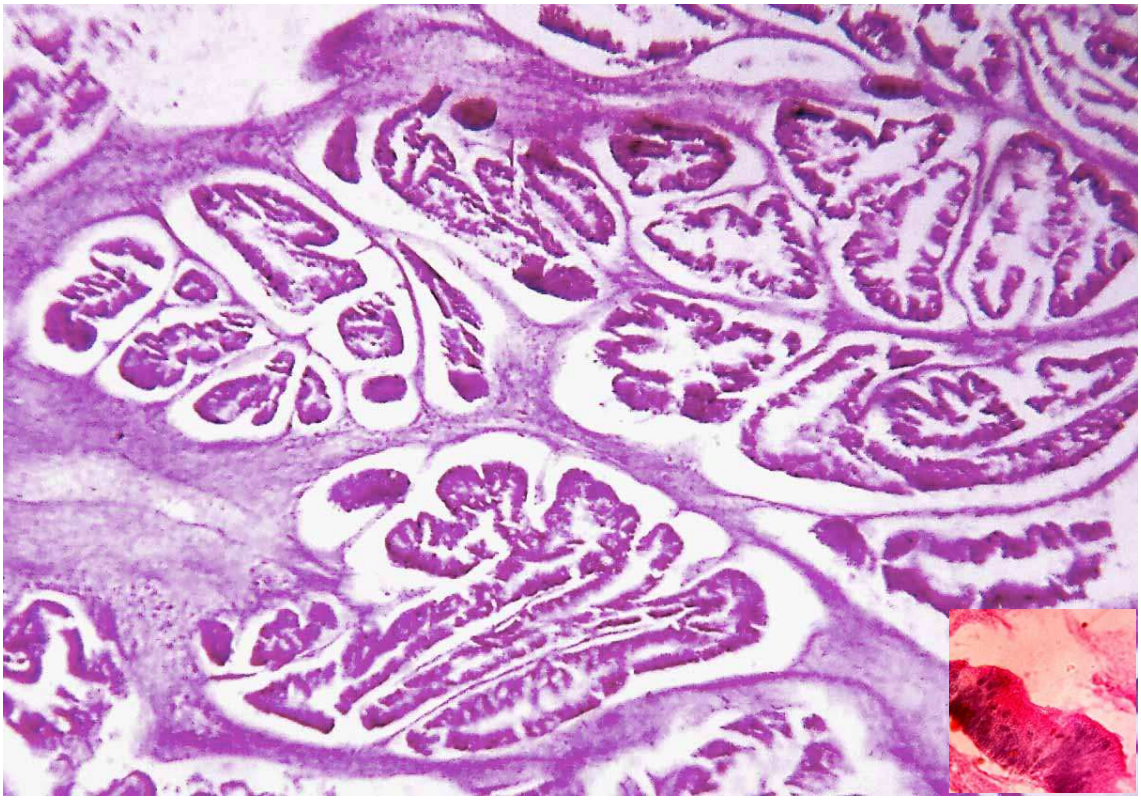

Figure 8 Mucinous cystadenocarcinoma- ***Photomicrograph shows tumour composed of irregular glands with back to back arrangement. Inset showing PAS Positivity (H&E 100 X) Inset (PAS 400 X)***

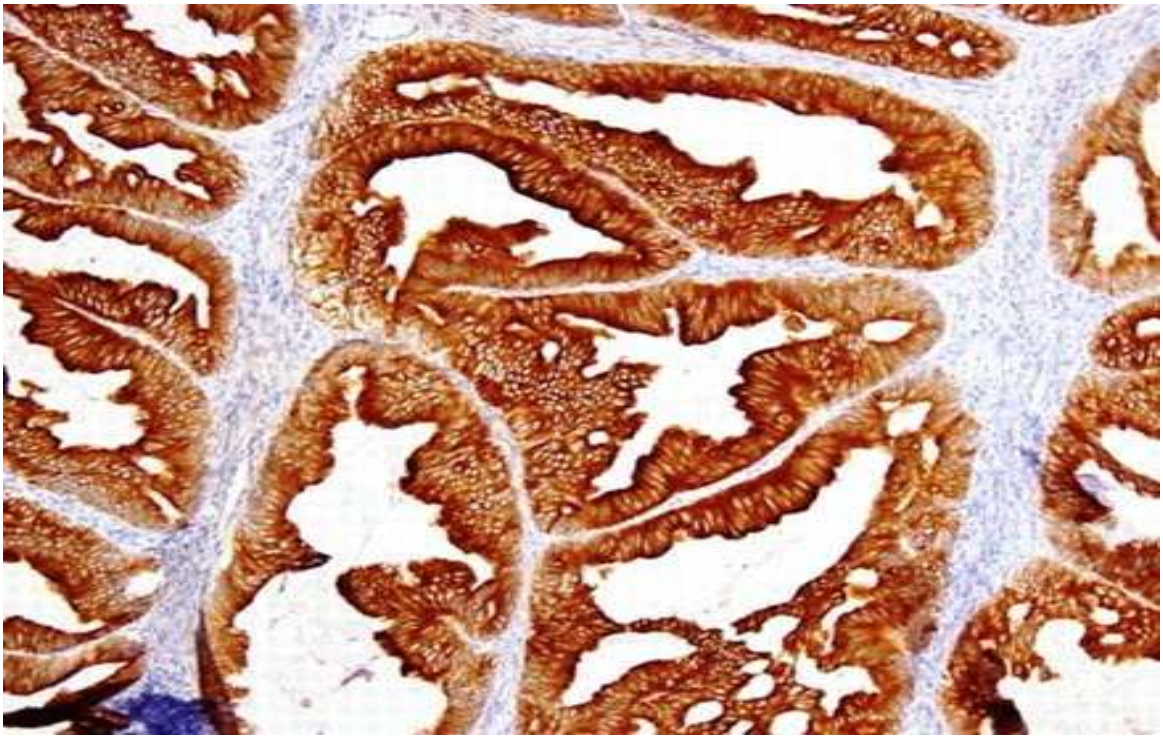

Figure 9 Mucinous cystadenocarcinoma-*Photomicrograph shows strong membrane positivity of tumour cells with CK 7. (Immunostain CK 7 100 X)*

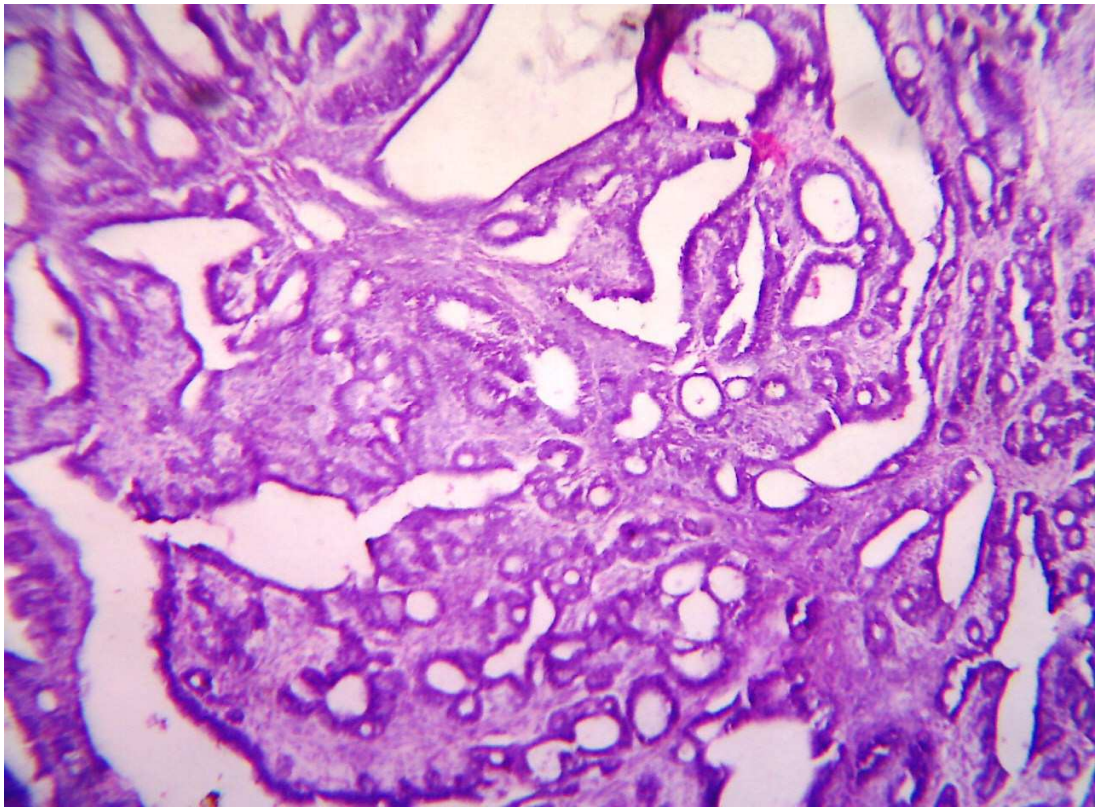

Figure 10 Endometrioid carcinoma- ***Photomicrograph showing tumour arranged in microglandular pattern lined by stratified columnar cells. (H&E 100 X)***

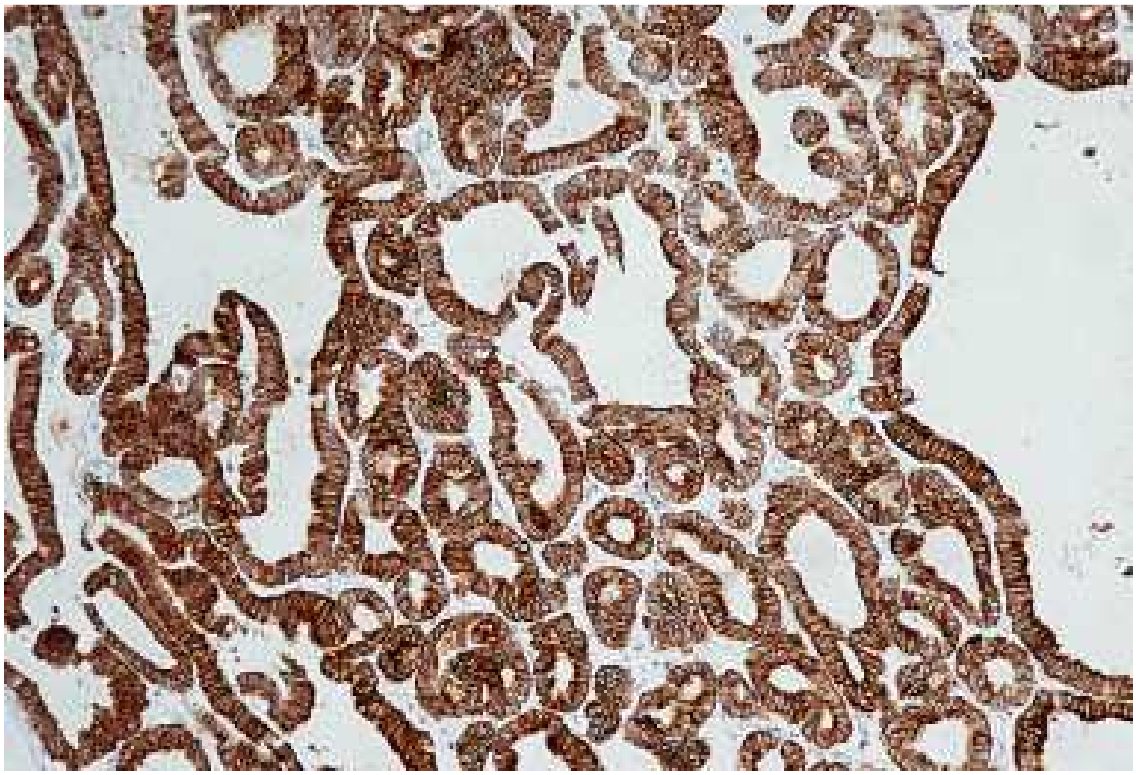

Figure 11 Endometrioid carcinoma-*Photomicrograph showing strong membrane positivity of tumour cells for Epithelial membrane antigen. (Immunostain EMA 100 X)*

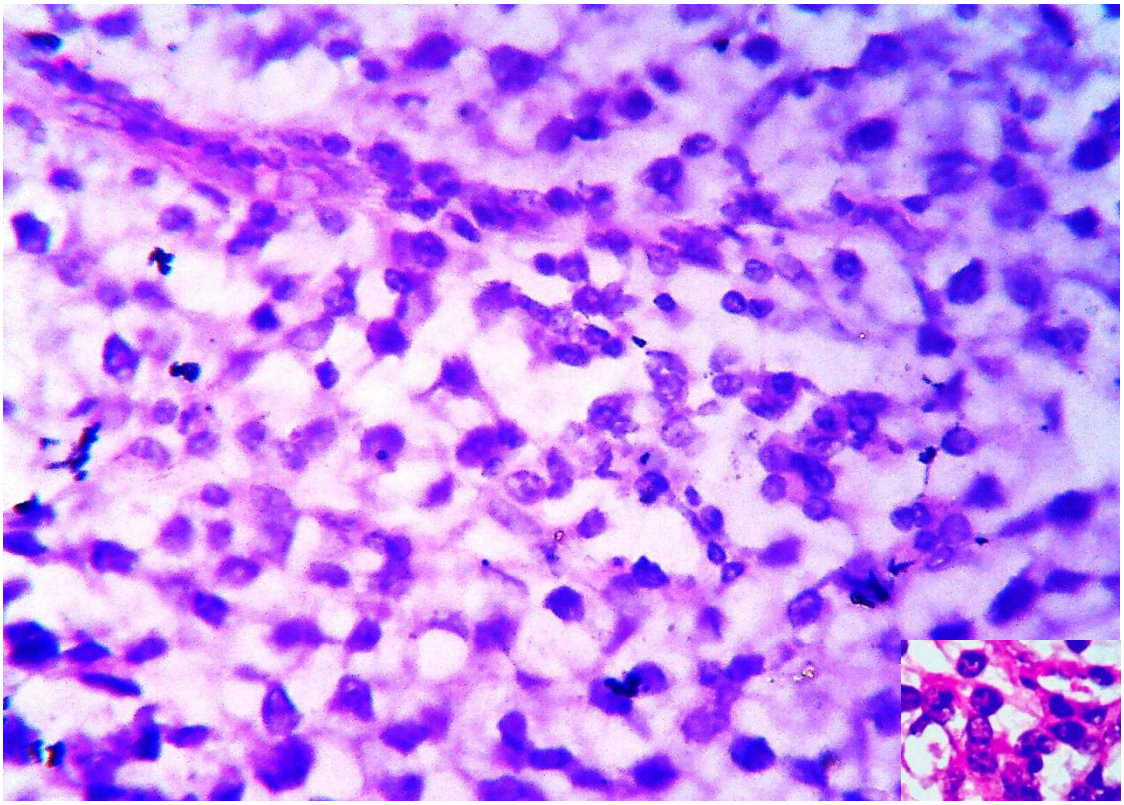

Figure 12 clear cell carcinoma-*Photomicrograph showing diffuse sheets of tumour cells, containing clear cytoplasm with irregular atypical nuclei. Inset showing Cytoplasmic PAS Positivity (H & E 100 X), Inset (PAS 400 X)*

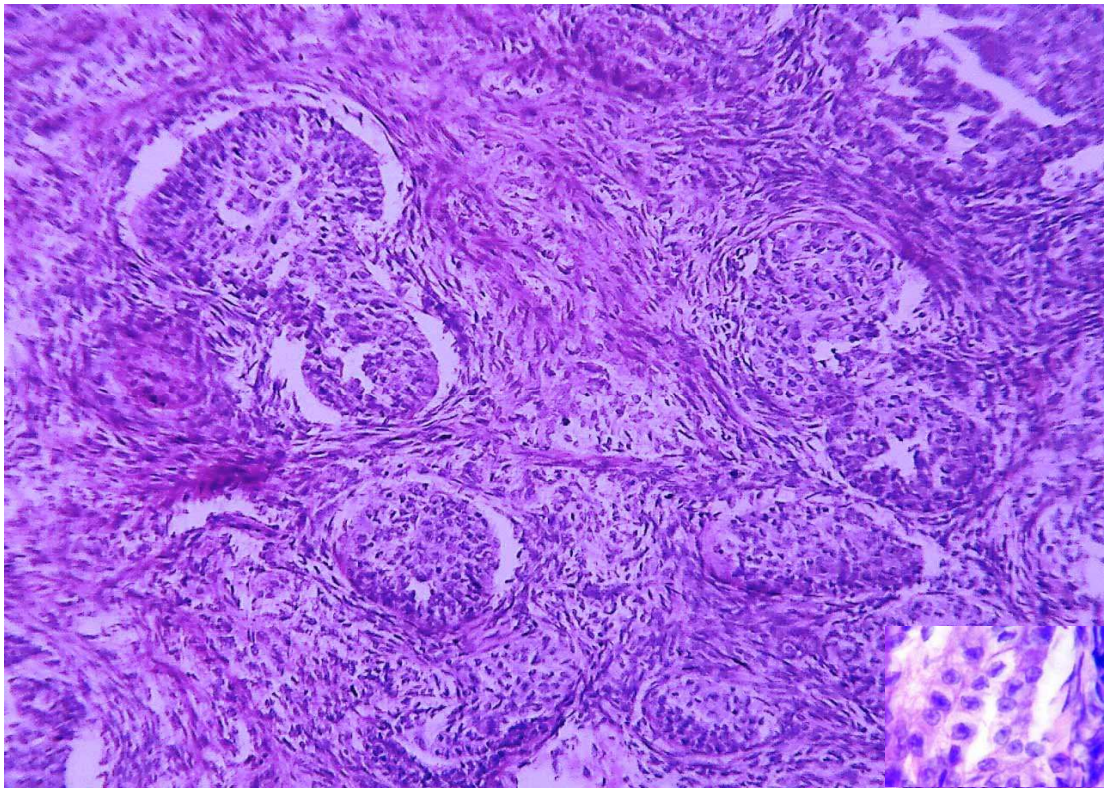

Figure 13 Brenner tumour-*Photomicrograph showing tumour, composed of solid nests of cells in a dense fibrous stroma. Inset showing longitudinal groove in the nuclei. (H & E 100 X) Inset (H&E 400 X)*

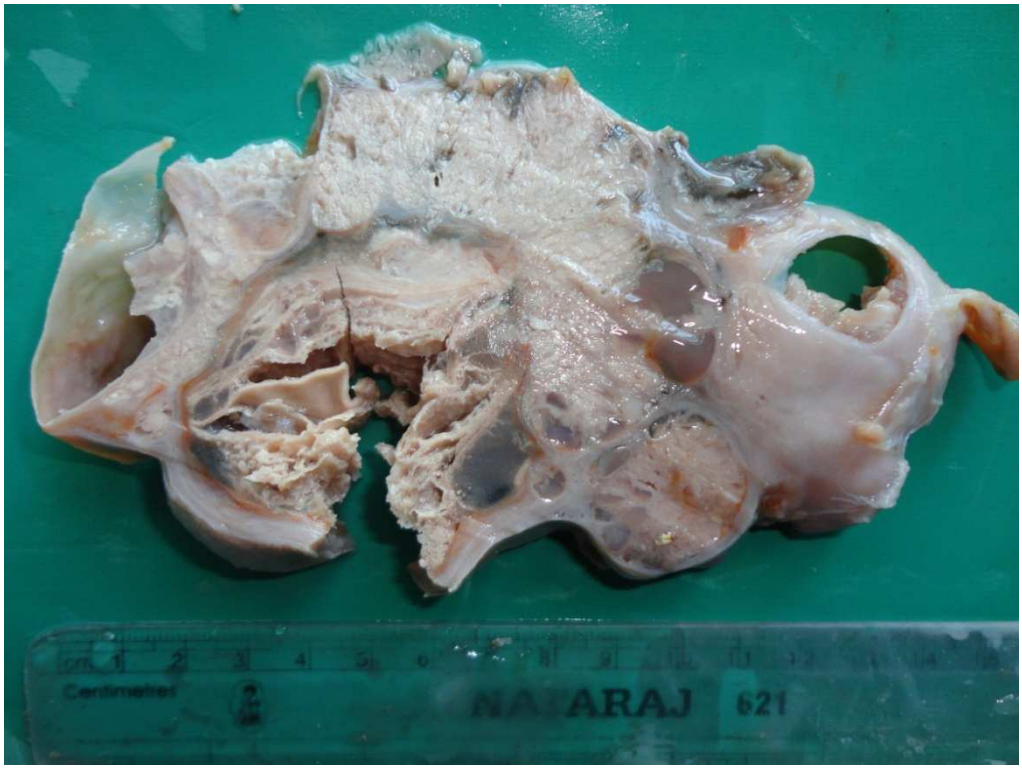

Figure 14 Granulosa cell tumour ***showing solid-cystic cut surface. Solid areas are greyish white to yellow in colour, admixed with thin walled cysts filled with mucoid material.***

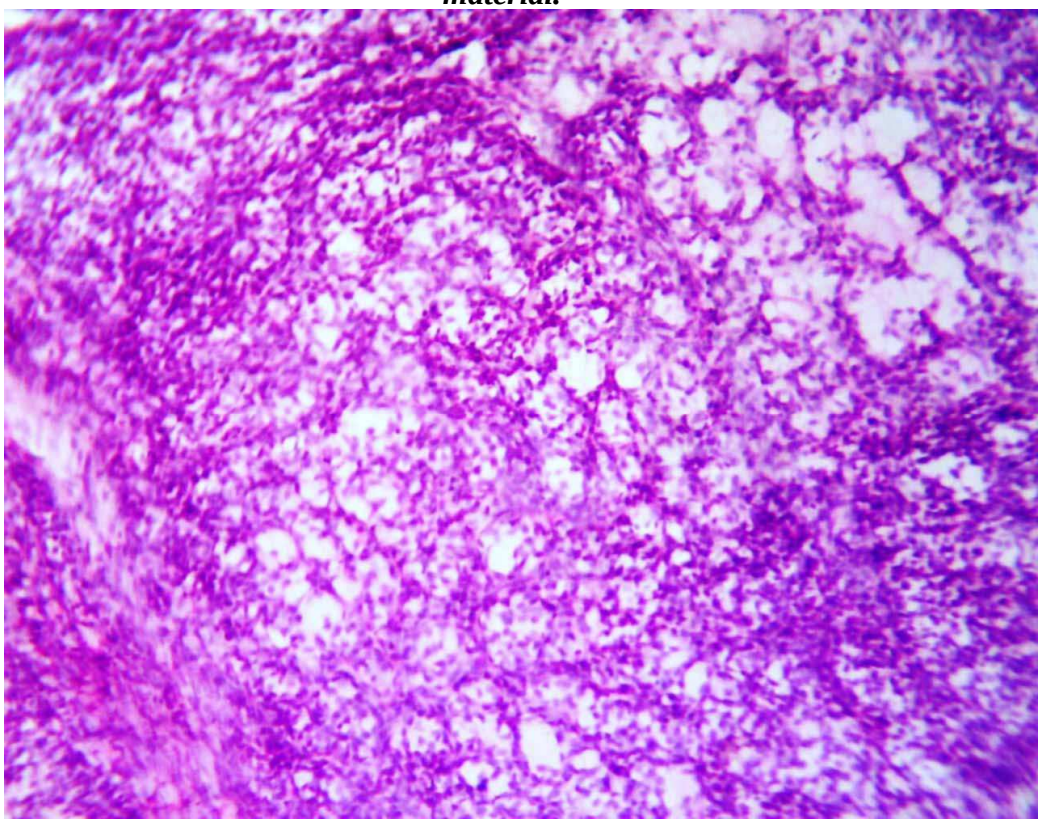

Figure 15 Granulosa cell tumour-*Photomicrograph showing tumour cells arranged in microfollicular pattern. (H & E 100X)*

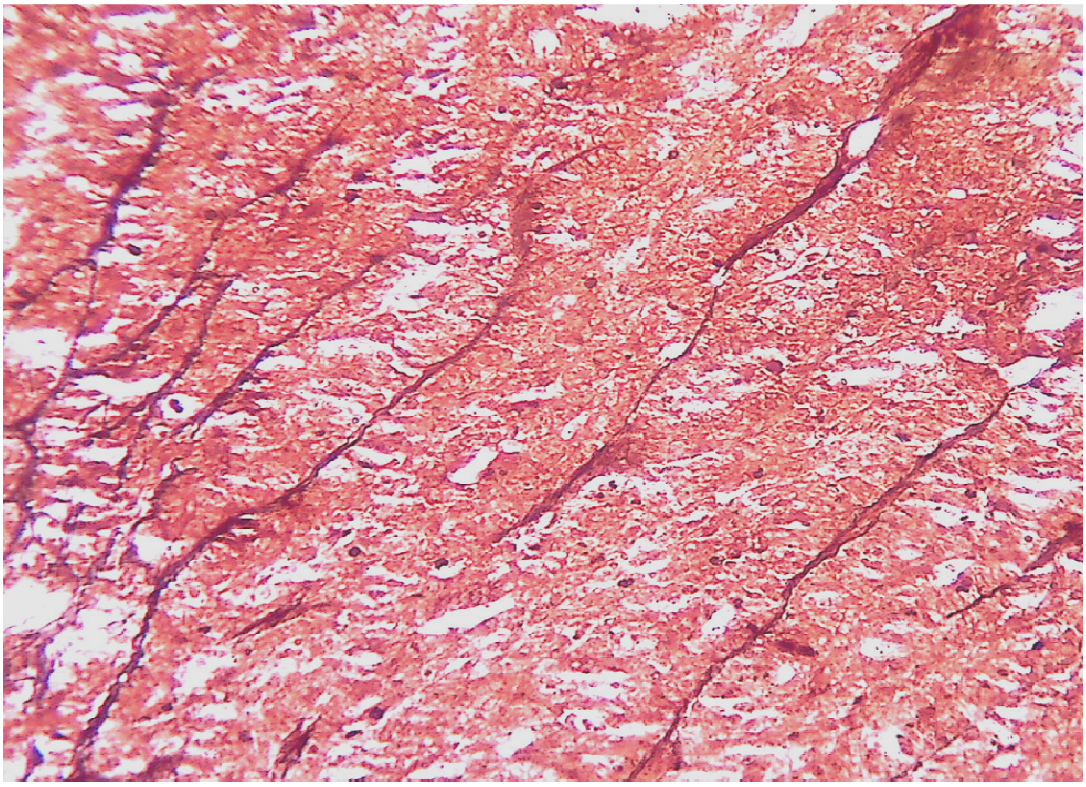

Figure 16 Granulosa cell tumour- ***Reticulin stain showing tumour cell clusters surrounded by reticulin fibres. (Reticulin stain 100 X)***

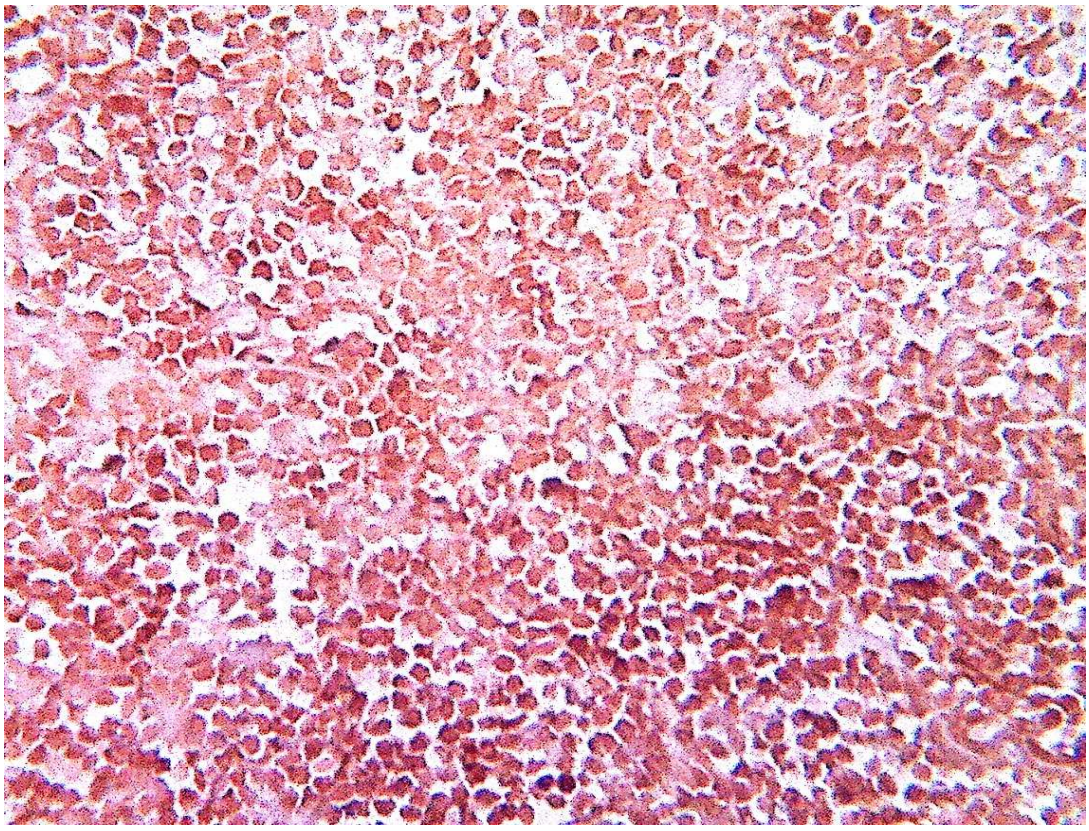

Figure 17 Granulosa cell tumour- ***Tumour cells exhibiting nuclear positivity with Alpha Inhibin (Immunostain-Inhibin 100 X)***

Figure 18 Fibrothecoma- ***Cut surface showing well circumscribed, uniformly solid tumour, greyish white in colour.***

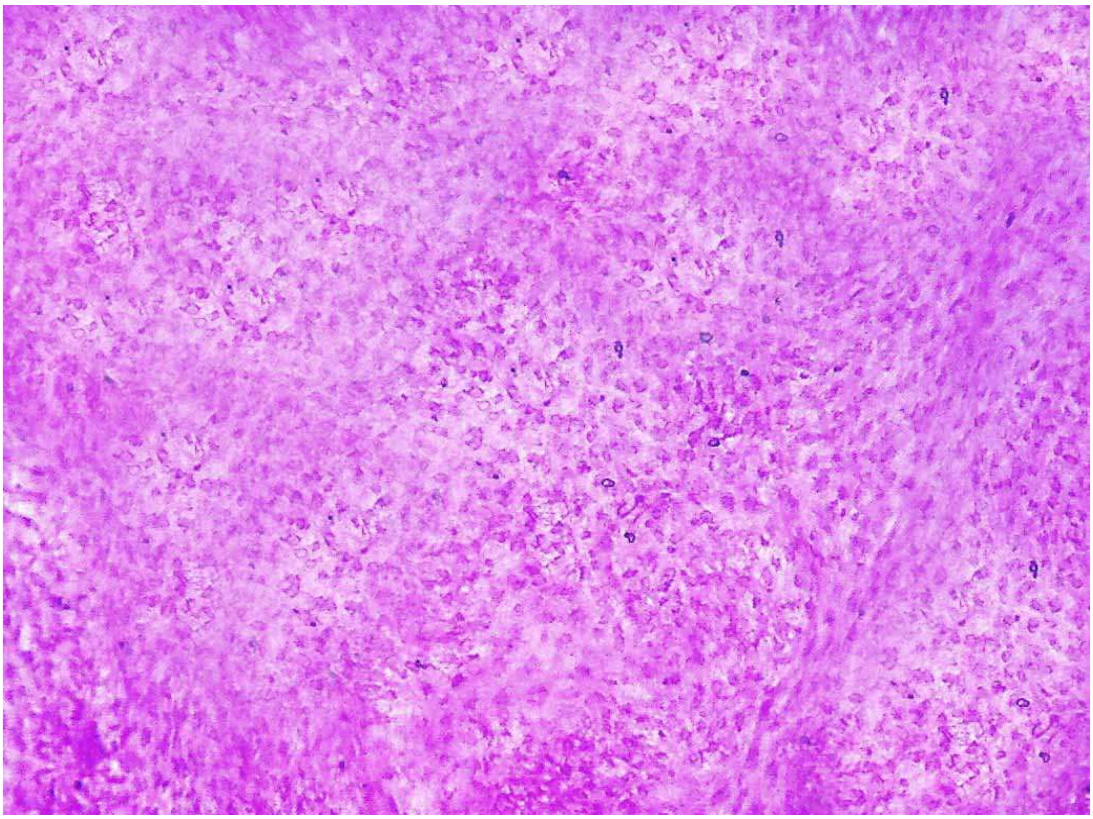

Figure 19 Fibrothecoma- ***Photomicrograph showing tumour composed of fascicles of plump spindle shaped cells (H & E 100 X)***

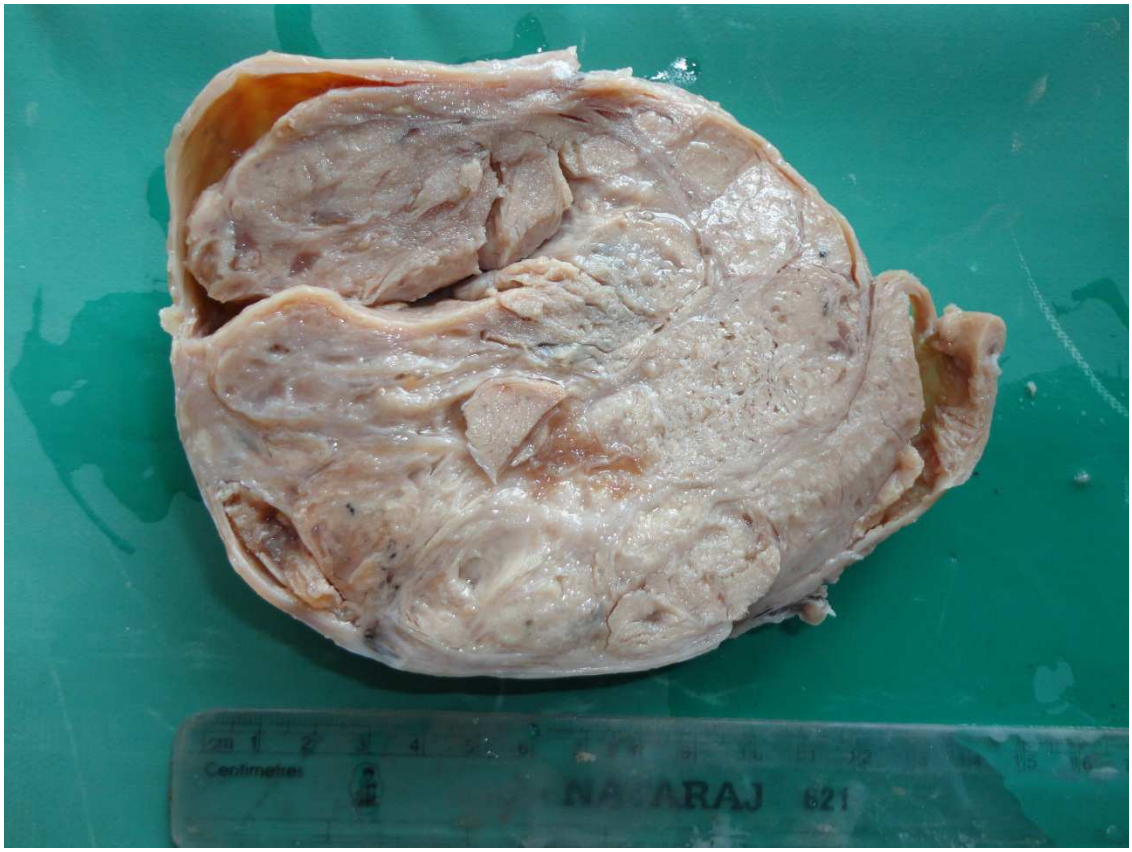

Figure 20 Malignant mixed germ cell tumour -*Cut surface showing solid variegated appearance.*

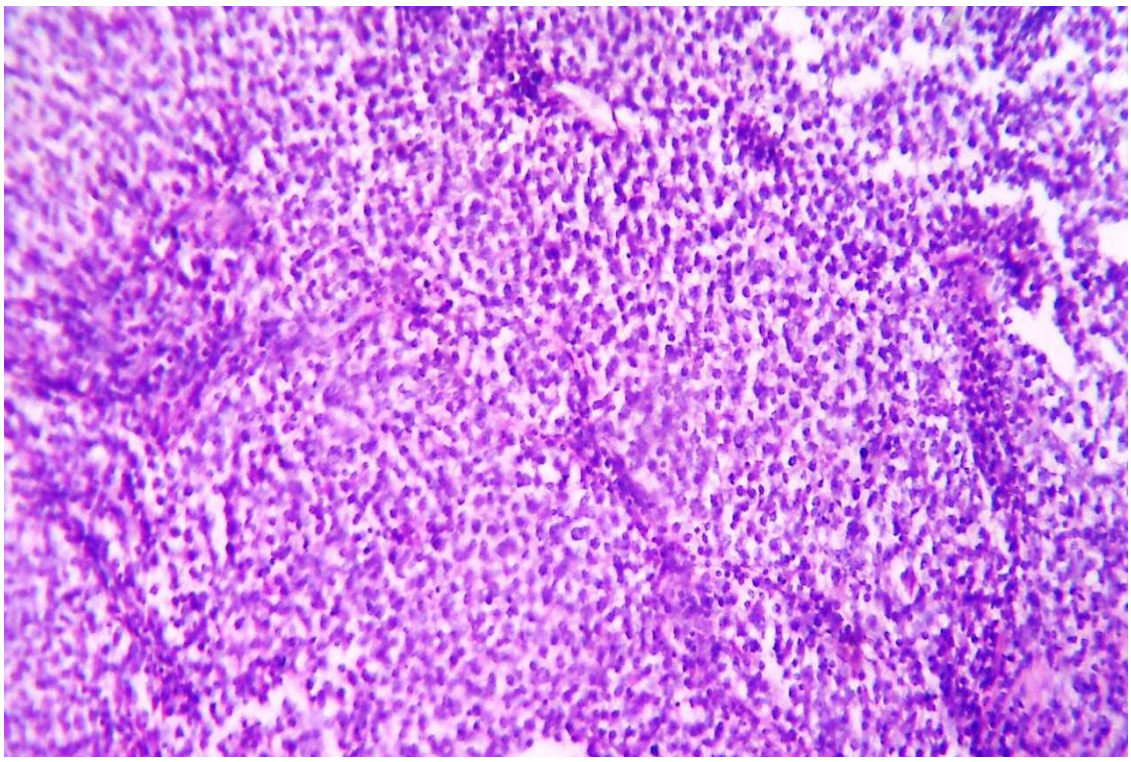

Figure 21 Dysgerminoma- *Photomicrograph showing solid sheets of tumour cells admixed with lymphocytic infiltration. (H & E 100 X)*

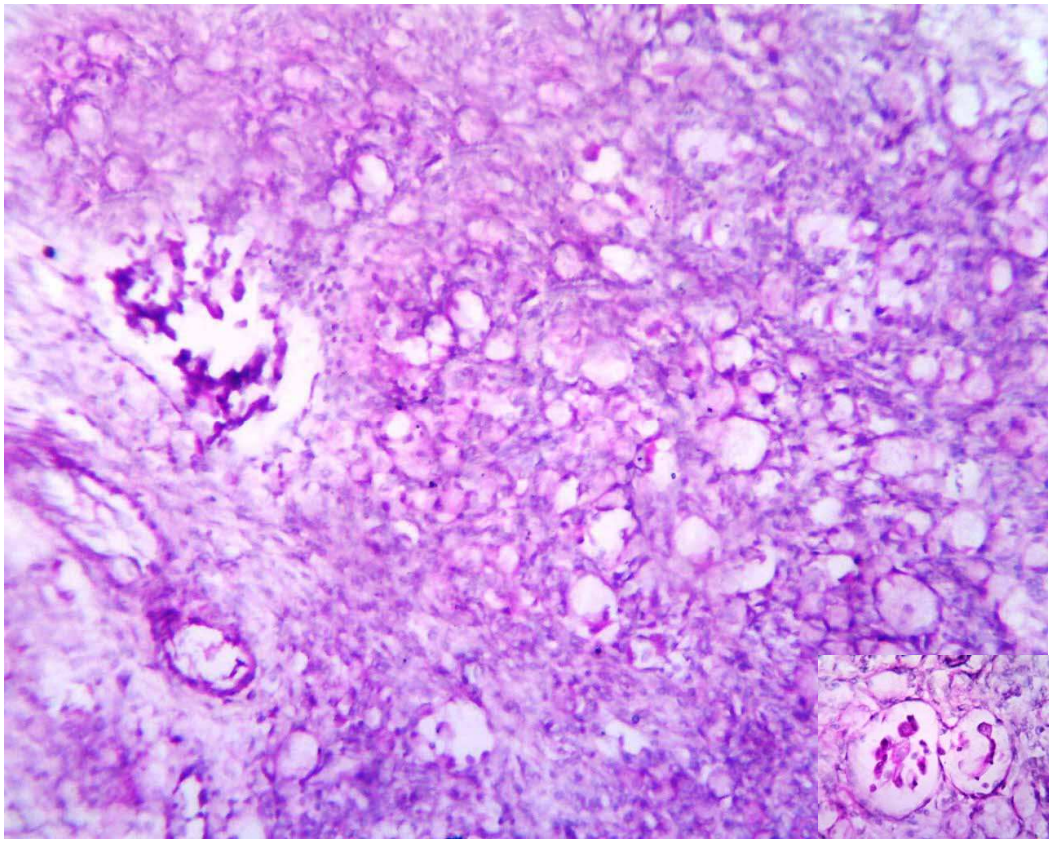

Figure 22 Yolk sac tumour- ***Photomicrograph showing tumour arranged in microcystic pattern. Inset showing Schiller Dual body (H & E 100 X) Inset (H & E 400 X)***

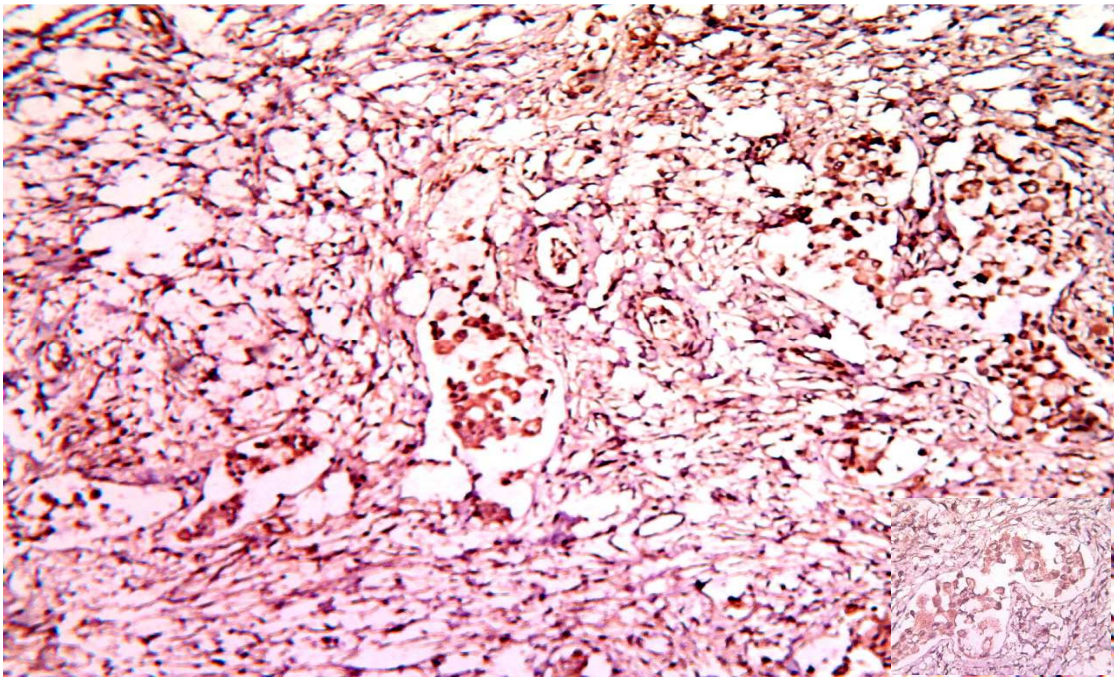

Figure 23 Yolk sac tumour ***exhibiting nuclear and cytoplasmic positivity with Alpha fetoprotein. Inset showing Schiller Dual body. (Immunostain AFP 100 X) Inset (Immunostain AFP 400 X)***

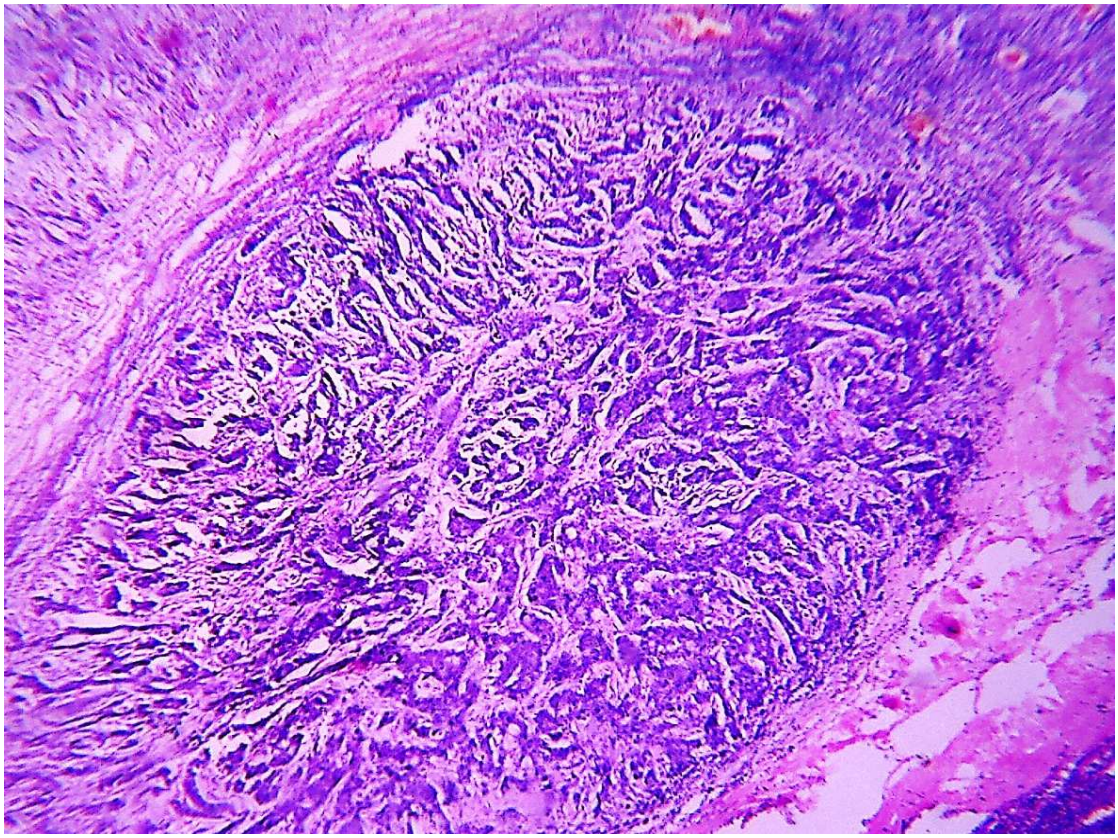

Figure 24 Ovarian Metastatic deposits- ***Photomicrograph showing tumour arranged in nodular infiltrative pattern composed of glandular structures. (H & E 40 X)***

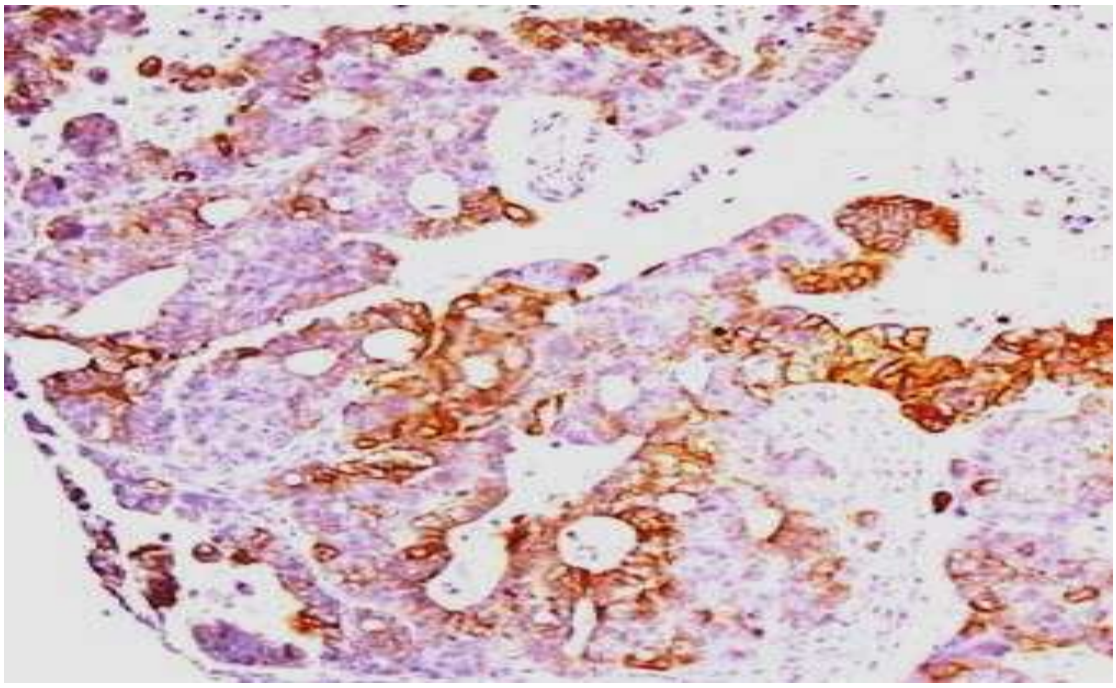

Figure 25 Ovarian Metastatic deposits –*Tumour cells exhibiting weak membrane positivity with CK 20. (Immunostain CK 20 100 X)*

Figure 26 Krukenberg tumours- ***Cut surface showing solid homogenous appearance, greyish white in colour.***

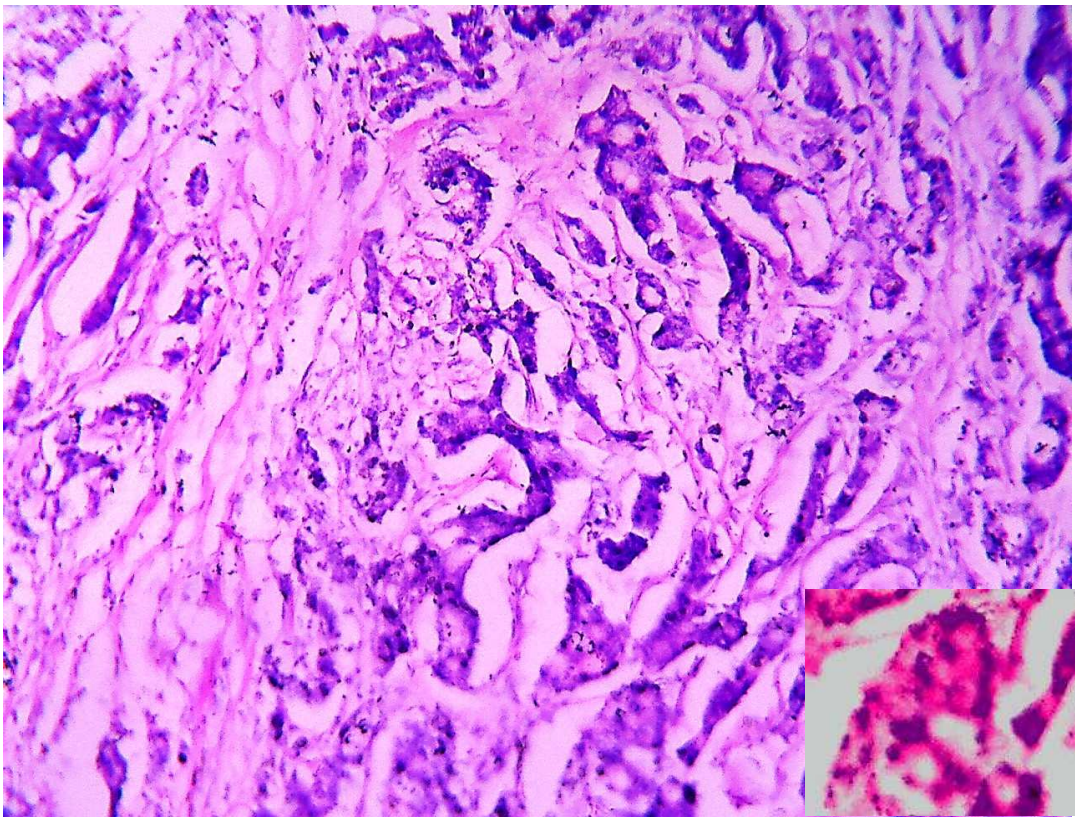

Figure 27 Krukenberg tumour- ***Photomicrograph showing tumour cells arranged in cords, nests composed of signet ring cells. Inset showing intracytoplasmic PAS positivity. (H & E 100 X) Inset (H & E 400 X)***

Figure 28 Massive edema of ovary –*Cut surface showing homogenous solid glistening appearance with peripheral rim of normal tissue.*

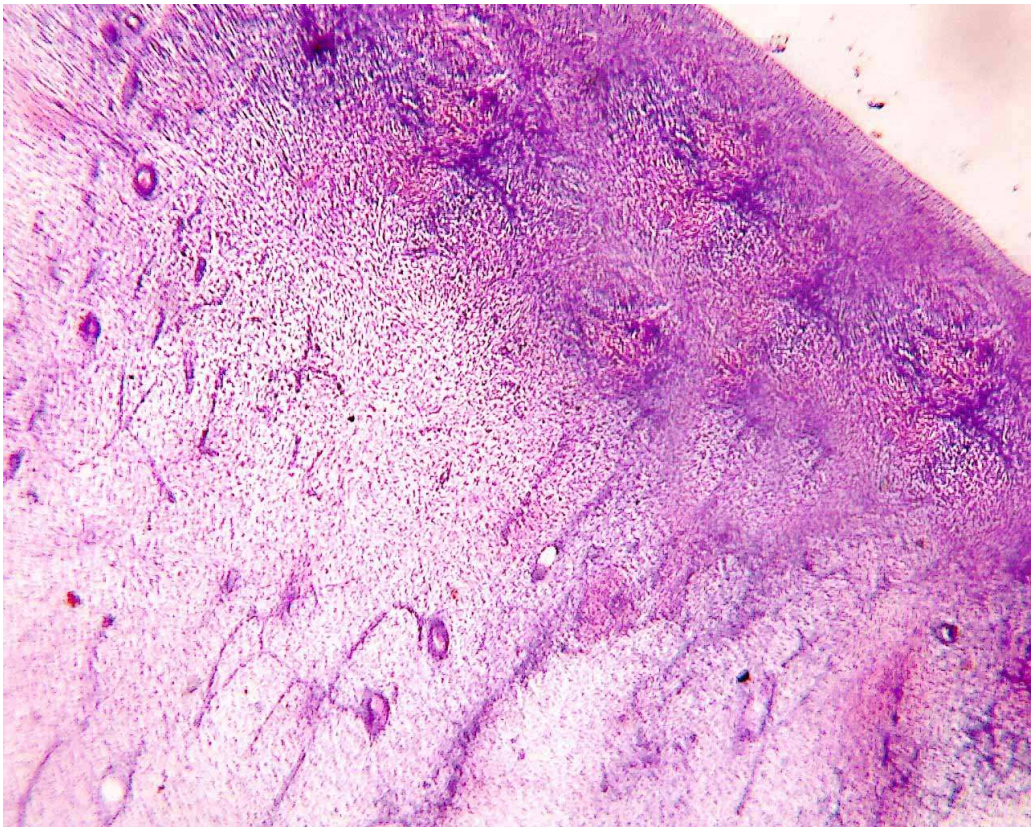

Figure 29 Massive edema of ovary –*Photomicrograph showing edematous ovarian stroma sparing the cortex. (H & E 100 X)*

S.No	IP No	HPE No	AGE(yrs)	PARITY		EXOGENOUS HORMONAL INTAKE		CLINICAL PRESENTATION			LATERALITY	GROSS				MICROSCOPIC DIAGNOSIS	OTHER STUDIES
				M	N	YES	NO	ABD.MASS	ABD PAIN	BLEEDING PV		SIZE(cms)	SOLID	CYSTIC	S & C		
1	46699	3 2008	27	M			NO		ABD PAIN		UL	5X5X2		C		MUCINOUS CYSTADENOMA	-
2	50011	13 2008	42	M			NO	ABD.MASS	ABD PAIN		UL	9X8X6			S & C	ENDOMETRIOTC CYST	-
3	46700	31 2008	51	M			NO		ABD PAIN		UL	9X4X3	S			SEROUS CYSTADENOCARCINOMA	-
4	46444	48 2008	43	M			NO		ABD PAIN		UL	11X7X3		C		SEROUS CYSTADENOMA	-
5	44976	52 2008	60	M			NO	ABD.MASS			UL	10X6X5		C		MUCINOUS CYSTADENOCARCINOMA	PAS-POSITIVE
6	50172	58 2008	65	M			NO	ABD.MASS	ABD PAIN		UL	19X14X7		C		MUCINOUS CYSTADENOMA	-
7	42740	70 2008	70	M			NO	ABD.MASS	ABD PAIN		UL	20X17X5		C		MUCINOUS CYSTADENOMA	PAS-POSITIVE
8	42748	137 2008	38	M			NO		ABD PAIN		UL	6X4X2		C		SEROUS CYSTADENOMA	-
9	46633	221 2008	40	M			NO		ABD PAIN		UL	5X3X2		C		DERMOID CYST	-
10	46870	228 2008	42	M			NO		ABD PAIN		UL	7X7X3		C		INCLUSION CYST	-
11	245	384 2008	38	M			NO	ABD.MASS	ABD PAIN		UL	6.5X5X2			S & C	SEROUS CYSTADENOMA	-
12	337	412 2008	23		N		NO	ABD.MASS			UL	8X5X3			S & C	ENDOMETRIOTIC CYST	-
13	876	435 2008	48	M			NO	ABD.MASS	ABD PAIN		UL	10X6X6			S & C	YOLK SAC TUMOR	AFP-Positive
14	1589	493 2008	33	M			NO		ABD PAIN		UL	7.5X5.5X4		C		MUCINOUS CYSTADENOMA	-
15	2939	524 2008	60	M			NO	ABD.MASS	ABD PAIN		UL	18X12X6			S & C	SEROUS CYSTADENOCARCINOMA	-
16	3042	561 2008	25	M		YES			ABD PAIN	BLEEDING PV	UL	5X5X2		C		SEROUS CYSTADENOMA	-
17	3378	683 2008	18		N		NO		ABD PAIN		UL	15X8X8	S			MIXED GERM CELL TUMOR	AFP-Positive
18	4890	742 2008	35	M			NO		ABD PAIN		UL	6X3X2		C		SEROUS CYSTADENOMA	-
19	4915	781 2008	22	M			NO		ABD PAIN		UL	7X4X3			S & C	INCLUSION CYST	-
20	5001	821 2008	52	M			NO	ABD.MASS	ABD PAIN		UL	10X9X7			S & C	ENDOMETRIOID CARCINOMA	EMA-Positive
21	5247	859 2008	45	M			NO		ABD PAIN	BLEEDING PV	UL	4X3X2		C		ENDOMETRIOTIC CYST	-
22	5377	935 2008	44	M			NO		ABD PAIN		UL	5X4X2	S			FIBROMA	-
23	5979	950 2008	45	M			NO			BLEEDING PV	UL	5X4X2		C		SEROUS CYSTADENOMA	-
24	6621	975 2008	38	M			NO		ABD PAIN		UL	5X2X1		C		SEROUS CYSTADENOMA	-
25	7731	1048 2008	25	M			NO		ABD PAIN		UL	11X5X5		C		SEROUS CYSTADENOMA	-
26	7924	1093 2008	50	M			NO	ABD.MASS	ABD PAIN		UL	30X25X5		C		MUCINOUS CYSTADENOMA	-
27	8612	1108 2008	45	M		YES			ABD PAIN		UL	6X3X2		C		SEROUS CYSTADENOMA	-
28	9960	1114 2008	37	M			NO		ABD PAIN		UL	10X6X5		C		MUCINOUS CYSTADENOMA	-
29	10007	1333 2008	45	M			NO	ABD.MASS	ABD PAIN		UL	15X4X5	S			FIBROTHECOMA	Reticulin-positive
30	10098	1347 2008	53	M			NO	ABD.MASS	ABD PAIN		UL	30X20X10			S&C	MUCINOUS CYSTADENOCARCINOMA	CK 7 positive &CK 20 negative
31	11003	1413 2008	77	M			NO		ABD PAIN		UL	6X5X3		C		INCLUSION CYST	-
32	21267	1446 2008	60	M			NO	ABD.MASS	ABD PAIN		BL	8X6X2	S			SEROUS CYSTADENOCARCINOMA	-
33	24350	1464 2008	40	M			NO		ABD PAIN		UL	5X3X2			S & C	SEROUS CYSTADENOFIBROMA	-
34	25000	1467 2008	40	M			NO			BLEEDING PV	UL	4X3.5X1		C		FOLLICLE CYST	-
35	26171	1509 2008	25		N		NO		ABD PAIN		UL	7X3X2		C		DERMOID CYST	-
36	23667	1538 2008	23	M			NO		ABD PAIN		UL	4X3X2		C		ENDOMETRIOTIC CYST	-
37	33803	1549 2008	29	M			NO		ABD PAIN		UL	6X5X4		C		DERMOID CYST	-
38	34515	1551 2008	25	M			NO		ABD PAIN		UL	6X5X4		C		DERMOID CYST	-
39	35804	1558 2008	33	M			NO		ABD PAIN		UL	9X6X4		C		MUCINOUS CYSTADENOMA	-
40	33383	1562 2008	44	M			NO		ABD PAIN		UL	6X5X5		C		SEROUS CYSTADENOMA	-
41	31004	1566 2008	33	M			NO		ABD PAIN		UL	6X5X5		C		MUCINOUS CYSTADENOMA	-
42	32417	1584 2008	39	M			NO	ABD.MASS	ABD PAIN		BL	18X15X10			S & C	SEROUS CYSTADENOCARCINOMA	-
43	34977	1622 2008	46		N		NO			BLEEDING PV	UL	6X4X3		C		SEROUS CYSTADENOMA	-
44	35072	1623 2008	37	M			NO			BLEEDING PV	BL	6X3X2		C		FOLLICLE CYST	-
45	34516	1692 2008	45	M			NO	ABD.MASS	ABD PAIN		UL	20X18X16		C		MUCINOUS CYSTADENOMA	-
46	38809	1718 2008	23	M			NO		ABD PAIN		UL	4X3X2		C		ENDOMETRIOTIC CYST	-
47	36962	1723 2008	38	M			NO		ABD PAIN		UL	7X5X4	S			TUBOOVARIAN ABSCESS	-
48	36950	1727 2008	31	M		YES			ABD PAIN		UL	5X2X2		C		ENDOMETRIOTIC CYST	-
49	37768	1741 2008	67	M			NO	ABD.MASS			UL	12X9X3		C		SEROUS CYSTADENOMA	-
50	39225	1842 2008	45	M			NO	ABD.MASS			UL	12X10X5		C		SEROUS CYSTADENOMA	-
51	41643	1857 2008	30	M			NO	ABD.MASS			UL	15X15X5		C		SEROUS CYSTADENOMA	-
52	47277	1858 2008	55	M			NO	ABD.MASS			BL	11X4X4	S			SEROUS CYSTADENOCARCINOMA	-
53	40733	1911 2008	44	M			NO	ABD.MASS			UL	2X11X8	S			FIBROTHECOMA	Reticulin-positive
54	42872	1962 2008	30	M			NO	ABD.MASS			UL	7X6X3		C		MUCINOUS CYSTADENOMA	-
55	45890	2020 2008	18	M			NO	ABD.MASS	ABD PAIN		UL	22X18X9	S			MIXED GERM CELL TUMOR	-
56	43557	2023 2008	68	M			NO	ABD.MASS			UL	15X9X8		C		SEROUS CYSTADENOMA	-
57	45745	2074 2008	38	M			NO	ABD.MASS	ABD PAIN		UL	35X30X10		C		MUCINOUS CYSTADENOMA	-
58	47826	2086 2008	40	M			NO	ABD.MASS	ABD PAIN		UL	14X8X2		C		SEROUS CYSTADENOMA	-
59	43079	2102 2008	48	M			NO	ABD.MASS			UL	5X4X3		C		SEROUS CYSTADENOMA	-
60	50749	2323 2008	27	M			NO	ABD.MASS			UL	7X6X5		C		SEROUS CYSTADENOMA	-
61	50677	2324 2008	30	M			NO	ABD.MASS			UL	20X13X7		C		MUCINOUS CYSTADENOMA	-
62	48027	2343 2008	52	M			NO	ABD.MASS	ABD PAIN		UL	10X10X5			S&C	STRUMA CARCINOID	-
63	50355	2344 2008	23	M			NO	ABD.MASS	ABD PAIN		UL	18X14X6		C		SEROUS CYSTADENOMA	-
64	41929	2345 2008	66	M			NO	ABD.MASS	ABD PAIN		BL	6X5X5	S			SEROUS CYSTADENOCARCINOMA-PD	WT 1 positive & CK 7 negative
65	51051	2380 2008	60	M			NO	ABD.MASS			UL	12X12X6		C		SEROUS CYSTADENOMA	-
66	222	109 2009	70	M			NO		ABD PAIN		UL	5x5x5		C		DERMOID CYST	-
67	1850	112 2009	22		N		NO	ABD.MASS			UL	12x8x4			S&C	SEROUS ADENOFIBROMA	-
68	53105	128 2009	58	M			NO	ABD.MASS			UL	8x6x3		C		SEROUS CYSTADENOMA	-

69	3839	222 2009	35	M			NO		ABD PAIN		UL	7X5X3		C		DERMOID CYST	-
70	4081	225 2009	42	M			NO		ABD PAIN		UL	7X5X3		C		SEROUS CYSTADENOMA	-
71	7151	240 2009	32	M			NO	ABD.MASS	ABD PAIN		UL	8X6X3		C		MUCINOUS CYSTADENOMA	-
72	5325	380 2009	27	M			NO		ABD PAIN		UL	7X6X4			S&C	DERMOID CYST	-
73	3034	385 2009	23	M			NO	ABD.MASS			UL	13X8X6			S&C	DERMOID CYST	-
74	9609	499 2009	32	M			NO	ABD.MASS			UL	16X9X4			S&C	SEROUS CYSTADENOMA	-
75	7954	509 2009	30	M			NO		ABD PAIN		UL	9x7x6		C		DERMOID CYST	-
76	10030	562 2009	19		N		NO	ABD.MASS	ABD PAIN		UL	11x10x5			S&C	DERMOID CYST	-
77	10538	642 2009	27	M			NO		ABD PAIN		UL	5x4x4		C		SEROUS CYSTADENOMA	-
78	10902	673 2009	65	M					ABD PAIN		BL	6X5X3			S&C	METASTATIC DEPOSITS	CK 7 Negative &CK 20 positive
79	10538	692 2009	47	M		YES				BLEEDING PV	UL	4X4X4		C		FOLLICULAR CYST	-
80	11079	786 2009	40	M			NO		ABD PAIN		UL	6X5X4		C		FOLLICULAR CYST	-
81	15808	860 2009	42	M			NO		ABD PAIN		UL	5X5X4			S&C	SEROUS CYSTADENO FIBROMA	-
82	16128	905 2009	43	M			NO		ABD PAIN		UL	5X3X3		C		FOLLICULAR CYST	-
83	16182	906 2009	42	M			NO	ABD.MASS	ABD PAIN		UL	17X15X8		C		SEROUS CYSTADENOMA	-
84	11883	924 2009	50	M			NO	ABD.MASS	ABD PAIN		UL	17X12X8		C		MUCINOUS CYSTADENOCARCINOMA	PAS-Positive
85	17095	937 2009	45	M			NO		ABD PAIN		BL	6X6X5		C		FOLLICULAR CYST	-
86	20789	1008 2009	25	M			NO		ABD PAIN		UL	7X6X4		C		SEROUS CYSTADENOMA	-
87	19837	1053 2009	40	M			NO		ABD PAIN		UL	5X4X3		C		SEROUS CYST	-
88	20154	1083 2009	32	M			NO	ABD.MASS	ABD PAIN		UL	10X6X4		C		ENDOMETRIOTIC CYST	-
89	18177	1095 2009	44	M			NO		ABD PAIN		UL	5X5X5		C		SEROUS CYSTADENOMA	-
90	24820	1239 2009	32	M			NO	ABD.MASS	ABD PAIN		UL	20X14X6		C		MUCINOUS CYSTADENOMA	-
91	29654	1300 2009	30	M			NO		ABD PAIN		UL	5X4X4			S&C	TUBOOVARIAN ABCESS	-
92	24559	1328 2009	55	M			NO		ABD PAIN		BL	7X7X5	S			SEROUS CYSTADENOCARCINOMA	-
93	23656	1410 2009	45	M			NO	ABD.MASS	ABD PAIN		UL	8X4X4	S			SEROUS CYSTADENOCARCINOMA	-
94	22136	1411 2009	50	M			NO	ABD.MASS			UL	10X5X5		C		MUCINOUS CYSTADENOMA	-
95	25350	1441 2009	26		N		NO		ABD PAIN		UL	6X4X4			S&C	ENDOMETRIOTIC CYST	-
96	28149	1473 2009	55	M			NO		ABD PAIN		UL	5X5X5		C		SEROUS CYSTADENOMA	-
97	25700	1476 2009	36	M			NO		ABD PAIN	BLEEDING PV	UL	7X6X6			S&C	GRANULOSA CELL TUMOR	Reticulin-positive,Alpha Inhibin positive
98	27889	1503 2009	42	M			NO		ABD PAIN		UL	5X4X4		C		SEROUS CYSTADENOMA	-
99	30639	1561 2009	30	M			NO		ABD PAIN		UL	5X4X4		C		LUTEAL CYST	-
100	28983	1635 2009	26	M			NO	ABD.MASS			UL	15X8X6		C		MUCINOUS CYSTADENOMA	-
101	30819	1641 2009	36	M			NO			BLEEDING PV	UL	5X4X3			S&C	ENDOMETRIOTIC CYST	-
102	28677	1642 2009	48	M			NO	ABD.MASS			UL	25X21X8		C		MUCINOUS CYSTADENOMA	-
103	32653	1806 2009	38	M			NO		ABD PAIN		UL	6X5X3		C		LUTEAL CYST	-
104	32297	1828 2009	24	M			NO		ABD PAIN		UL	7X5X3		C		ENDOMETRIOTIC CYST	-
105	32628	1865 2009	48	M			NO		ABD.PAIN		BL	5X5X4			S&C	FOLLICULAR CYST	-
106	31633	1866 2009	50	M			NO	ABD.MASS	ABD.PAIN		UL	5X4X4		C		DERMOID CYST	-
107	34069	1912 2009	55	M			NO	ABD.MASS	ABD.PAIN		UL	20X10X8			S&C	CLEAR CELL ADENOCARCINOMA	PAS-Positive
108	32797	1918 2009	65	M			NO		ABD.PAIN		UL	5X4X3			S&C	SEROUS CYSTADENO FIBROMA	-
109	29936	1978 2009	47	M			NO	ABD.MASS	ABD.PAIN		UL	6X6X6			S&C	MIXED GERM CELL TUMOR	AFP-Positive
110	31322	1983 2009	50	M			NO		ABD.PAIN		UL	6X3X3		C		SEROUS CYSTADENO FIBROMA	-
111	34955	1986 2009	47	M			NO		ABD.PAIN	BLEEDING PV	UL	5X4X3		C		ENDOMETRIOTIC CYST	-
112	34948	2047 2009	50	M			NO	ABD.MASS			UL	15X10X5		C		MUCINOUS CYSTADENOMA	-
113	34627	2061 2009	24		N		NO		ABD.PAIN		BL	7X4X3		C		DERMOID CYST	-
114	36635	2062 2009	35	M			NO	ABD.MASS	ABD.PAIN		BL	18X14X4			S&C	SEROUS CYSTADENOCA	-
115	34618	2093 2009	66	M			NO	ABD.MASS	ABD.PAIN		UL	10X10X5		C		DERMOID CYST	-
116	37263	2104 2009	45	M			NO	ABD.MASS	ABD.PAIN		UL	28X21X11		C		SEROUS CYSTADENOMA	-
117	37652	2137 2009	45	M			NO		ABD.PAIN		UL	5X5X2		C		MUCINOUS CYSTADENOMA	-
118	36934	2245 2009	39	M			NO		ABD.PAIN	BLEEDING PV	UL	6X4X4			S&C	ENDOMETRIOTIC CYST	-
119	37648	2271 2009	45	M			NO		ABD.PAIN		UL	6X4X3		C		SEROUS CYSTADENOMA	-
120	39358	2272 2009	38	M			NO		ABD.PAIN		UL	6X4X4			S&C	ENDOMETRIOTIC CYST	-
121	41895	2297 2009	60	M			NO	ABD.MASS	ABD.PAIN		UL	12X9X9	S			SEROUS CYSTADENOCARCINOMA-PD	WT-1 positive & CK 7positive
122	39877	2305 2009	35	M			NO	ABD.MASS			UL	15X8X5	S			FIBROTHECOMA	Reticulin-positive
123	55271	2309 2009	33	M			NO	ABD.MASS			UL	10X4X4		C		SEROUS CYSTADENOMA	-
124	41541	2368 2009	60	M			NO	ABD.MASS	ABD.PAIN		UL	10X8X8		C		MUCINOUS CYSTADENOMA	-
125	39883	2423 2009	66	M			NO	ABD.MASS			UL	10X5X5		C		MUCINOUS CYSTADENOMA	-
126	44345	2502 2009	67	M			NO	ABD.MASS	ABD.PAIN		UL	9X5X3			S&C	SEROUS CYSTADENO CARCINOMA	-
127	44648	2512 2009	27	M			NO	ABD.MASS	ABD.PAIN		UL	13X8X4	S			SEROUS CYSTADENOCARCINOMA	-
128	45300	2542 2009	32	M			NO		ABD.PAIN		UL	6X4X4		C		SEROUS CYSTADENOMA	-
129	46653	2555 2009	60	M			NO	ABD.MASS			UL	10X10X5		C		MUCINOUS CYSTADENOMA	-
130	45636	2575 2009	51	M			NO		ABD.PAIN		UL	5X4X4		C		DERMOID CYST	-
131	46129	2691 2009	30	M			NO	ABD.MASS		BLEEDING PV	UL	8X8X7		C		ENDOMETRIOTIC CYST	-
132	57590	2754 2009	37	M			NO	ABD.MASS			UL	10X6X6		C		MUCINOUS CYSTADENOMA	-
133	50237	61 2010	32	M			NO		ABD.PAIN		UL	6X6X5		C		ENDOMETRIOTIC CYST	-
134	54032	108 2010	70	M			NO	ABD.MASS	ABD.PAIN		UL	8X7X7			S&C	SEROUS CYSTADENO CA	-
135	52648	115 2010	27	M			NO		ABD.PAIN		UL	7X4X4		C		ENDOMETRIOTIC CYST	-
136	54958	140 2010	25	M			NO	ABD.MASS	ABD.PAIN		UL	11X9X4		C		SEROUS CYSTADENOMA	-
137	1140	197 2010	15		N		NO		ABD.PAIN		BL	6X4X4			S&C	DERMOID CYST	-
138	3989	217 2010	42	M			NO		ABD.PAIN		UL	8X4X4		C		SEROUS CYSTADENOMA	-
139	2748	271 2010	27	M			NO	ABD.MASS	ABD.PAIN		UL	12X7X5			S&C	ENDOMETRIOTIC CYST	-

140	6549	307 2010	23		N		NO		ABD.PAIN		UL	6X3X3		C		SEROUS CYSTADENOMA	-
141	6713	310 2010	32	M			NO	ABD.MASS	ABD.PAIN		UL	10X6X3		C		ENDOMETRIOTIC CYST	-
142	2350	331 2010	40	M			NO	ABD.MASS			UL	18X10X8		C		MUCINOUS CYSTADENOMA	-
143	7545	353 2010	27	M			NO	ABD.MASS			UL	10X7X6		C		SEROUS CYSTADENOMA	-
144	6213	399 2010	50	M			NO		ABD.PAIN		UL	9X6X3		C		DERMOID CYST	-
145	10907	513 2010	21		N		NO	ABD.MASS	ABD.PAIN		UL	22X16X8			S&C	MIXED GERM CELL TUMOR	AFP-Positive
146	10900	514 2010	30	M		YES			ABD.PAIN	BLEEDING PV	UL	7X5X2		C		FOLLICULAR CYST	-
147	8832	528 2010	38	M			NO	ABD.MASS	ABD.PAIN		BL	13X12X8		C		MUCINOUS CYSTADENOMA	-
148	9009	533 2010	40	M			NO		ABD.PAIN		UL	4X3X3			S&C	ENDOMETRIOTIC CYST	-
149	11539	555 2010	45	M			NO	ABD.MASS	ABD.PAIN		UL	14X9X5	S			SEROUS CYSTADENOCARCINOMA-PD	WT-1 positive & CK 7positive
150	14364	592 2010	25		N		NO	ABD.MASS	ABD.PAIN		UL	9X4X4			S&C	DERMOID CYST	-
151	11306	620 2010	42	M			NO	ABD.MASS	ABD.PAIN		UL	16X9X5		C		SEROUS CYSTADENOMA	-
152	11470	627 2010	25	M			NO	ABD.MASS	ABD.PAIN		UL	20X18X5		C		MUCINOUS CYSTADENOMA	-
153	14465	636 2010	45	M			NO	ABD.MASS			UL	5X5X5		C		SEROUS CYSTADENOMA	-
154	12212	710 2010	35	M			NO	ABD.MASS			UL	5X5X4		C		SEROUS CYSTADENOMA	-
155	15619	747 2010	66	M			NO	ABD.MASS	ABD.PAIN		BL	6X5X5			S&C	MUCINOUS CYSTADENO CARCINOMA	CK 7 positive &CK 20 negative
156	14882	758 2010	54	M			NO	ABD.MASS	ABD.PAIN		UL	10X8X8		C		MUCINOUS CYSTADENOMA	-
157	12392	764 2010	55	M			NO	ABD.MASS	ABD.PAIN		UL	6X6X5		C		SEROUS CYSTADENOMA	-
158	17270	765 2010	27	M			NO	ABD.MASS			UL	7X5X4		C		DERMOID CYST	-
159	17816	782 2010	30	M			NO	ABD.MASS	ABD.PAIN		UL	19X12X6		C		SEROUS CYSTADENOMA-BL	-
160	18350	809 2010	27	M			NO	ABD.MASS	ABD.PAIN		UL	14X9X5		C		SEROUS CYSTADENOMA	-
161	18836	872 2010	47	M			NO	ABD.MASS	ABD.PAIN		BL	9X5X5	S			KRUKENBERG TUMOR	PAS-Positive
162	20507	986 2010	40	M			NO	ABD.MASS	ABD.PAIN		BL	11X5X5	S			SEROUS CYSTADENOCARCINOMA-PD	WT-1 positive & CK 7 Negative
163	23461	987 2010	40	M			NO	ABD.MASS			UL	12X11X4		C		SEROUS CYSTADENOMA	-
164	23415	1024 2010	54	M			NO	ABD.MASS	ABD.PAIN		UL	22X19X10			S&C	GRANULOSA CELL TUMOUR	Reticulin-positive,Alpha Inhibin- positive
165	25141	1027 2010	20		N		NO	ABD.MASS	ABD.PAIN		UL	14X11X4		C		SEROUS CYSTADENOMA	-
166	22439	1056 2010	65	M			NO	ABD.MASS	ABD.PAIN		UL	6X6X6			S&C	SEROUS CYSTADENOCARCINOMA-PD	WT-1 positive & CK 7positive
167	19570	1082 2010	67	M			NO	ABD.MASS	ABD.PAIN		UL	11X10X7	S			BRENNER TUMOUR	-
168	20181	1145 2010	52	M			NO	ABD.MASS			UL	9X6X6	S			FIBROMA	-
169	28213	1156 2010	40	M			NO	ABD.MASS	ABD.PAIN		UL	11X8X4		C		SEROUS CYSADENOMA	-
170	27725	1157 2010	25	M			NO	ABD.MASS			UL	18X6X2		C		SEROUS CYSTADNOMA	-
171	26380	1196 2010	52	M			NO	ABD.MASS	ABD.PAIN		UL	18X10X5		C		MUCINOUS CYSTADENOMA	-
172	28559	1205 2010	62	M			NO	ABD.MASS	ABD.PAIN		BL	11X8X7			S&C	SEROUS CYSATADENOCARCINOMA	-
173	27004	1223 2010	27	M			NO	ABD.MASS			UL	12X6X6		C		LUTEAL CYST	-
174	26834	1233 2010	50	M			NO	ABD.MASS	ABD.PAIN		UL	19X18X6			S&C	SEROUS CYSTADENOCARCINOMA	-
175	30494	1285 2010	52	M			NO	ABD.MASS	ABD.PAIN		UL	24X18X8		C		MUCINOUS CYSTADENOMA	-
176	28021	1340 2010	50	M			NO	ABD.MASS	ABD.PAIN		UL	20X17X10		C		MUCINOUS CYSTADENOMA	-
177	30513	1377 2010	52	M			NO	ABD.MASS	ABD.PAIN		UL	12X7X5		C		SEROUS CYSTADENOMA	-
178	30417	1390 2010	70	M			NO	ABD.MASS		BLEEDING PV	UL	12X11X8			S&C	ENDOMETRIOTIC CYST	-
179	31518	1449 2010	40	M			NO		ABD.PAIN		UL	5X4X4		C		SEROUS CYSTADENOMA	-
180	31750	1471 2010	43	M			NO		ABD.PAIN		UL	5X2X2		C		DERMOID CYST	-
181	32158	1479 2010	27	M			NO	ABD.MASS	ABD.PAIN		UL	9X5X5		C		ENDOMETRIOTIC CYST	-
182	34162	1509 2010	38	M			NO		ABD.PAIN		UL	4X2X2		C		LUTEAL CYST	-
183	33860	1532 2010	55	M			NO	ABD.MASS	ABD.PAIN		UL	18X12X6			S&C	SEROUS-BL TUMOR	-
184	36277	1888 2010	50	M			NO		ABD.PAIN		UL	5X3X3		C		SEROUS CYSTADENOMA	-
185	38600	1898 2010	53	M			NO		ABD.PAIN		UL	6X4X2		C		SEROUS CYSTADENOMA	-
186	41423	2092 2010	40	M			NO		ABD.PAIN		UL	7X4X3		C		SEROUS CYSTADENOMA	-
187	42535	2093 2010	35	M			NO		ABD.PAIN		UL	7X6X5		C		SEROUS CYSTADENOMA	-
188	43826	2139 2010	66	M			NO	ABD.MASS	ABD.PAIN		BL	8X6X5			S &C	MUCINOUS CYSTADENOCARCINOMA	PAS-Positive,CK 7 positive &CK 20 negative
189	46182	2144 2010	45	M			NO	ABD.MASS	ABD.PAIN		UL	12X7X7		C		SEROUS CYSTADENOMA	-
190	43100	2204 2010	37	M			NO	ABD.MASS	ABD.PAIN		UL	12X9X3		C		SEROUS CYSTADENOMA	-
191	46982	2272 2010	23		N		NO		ABD.PAIN		UL	7X6X5		C		SEROUS CYSTADENOMA	-
192	49383	2304 2010	15		N		NO		ABD.PAIN		UL	7X5X4		C		DERMOID CYST	-
193	46547	2320 2010	66	M			NO	ABD.MASS	ABD.PAIN		BL	10X5X5			S&C	SEROUS CYSTADENOCARCINOMA-PD	WT-1 positive & CK 7positive
194	46991	2387 2010	48	M			NO	ABD.MASS	ABD.PAIN		BL	20X16X10			S&C	MUCINOUS CYSTADENOCARCINOMA	PAS-positive,CK 7 positive &CK 20 negative
195	51720	2403 2010	30	M			NO	ABD.MASS	ABD.PAIN		UL	9X7X6		C		DERMOID CYST	-
196	51680	2500 2010	24	M			NO		ABD.PAIN		UL	5X5X4		C		SEROUS CYSTADENOMA	-
197	50731	2537 2010	32	M			NO		ABD.PAIN		UL	7X5X5		C		SEROUS CYSTADENOMA	-
198	52896	2580 2010	47	M			NO	ABD.MASS	ABD.PAIN		UL	9X6X4		C		SEROUS CYSTADENOMA	-
199	57567	2581 2010	49	M			NO	ABD.MASS	ABD.PAIN		UL	8X7X5	S			SEROUS CYSTADENOCARCINOMA-PD	WT-1 positive & CK 7positive
200	54251	2592 2010	60	M			NO	ABD.MASS	ABD.PAIN		UL	17X6X4		C		MUCINOUS CYSTADENOMA	-
201	52282	2611 2010	45	M			NO		ABD.PAIN		UL	9X7X3		C		DERMOID CYST	-
202	53929	2621 2010	27	M			NO		ABD.PAIN		UL	7X4X2		C		SEROUS CYSTADENOMA	-
203	56058	2622 2010	40	M			NO		ABD.PAIN		UL	7X6X4		C		SEROUS CYSTADENOMA	-

APPENDIX I

PROFORMA

NAME:

DATE:

AGE:

I.P NO/O.P NO:

ADDRESS:

BIOPSY NO:

CLINICAL HISTORY:

- Presenting complaints-
- Menstrual history-
- Marital history-
- Parity-
- Past history-

  H/O exogenous hormonal intake

  Previous clinical illness/surgery
- Family history

GENERAL EXAMINATION

- Anaemia
- Jaundice
- Pedal edema
- Generalised lymphadenopathy

SYSTEMIC EXAMINATION

- CVS
- RS
- ABDOMEN

INVESTGATIONS:

Blood investigations:

- Hb
- Serum markers-CA-125, AFP, HCG, Lipid profile, Thyroid function test

Radiological examinations

- USG -
- CT -
- MRI -

FNAC FINDINGS:

CLINICAL DIAGNOSIS:

SURGERY DONE:

HISTOPATHOLOGICAL EXAMINATION:

- Gross features

- Microscopic features

- Other studies
  - ✓ Special stain-
  - ✓ Immunohistochemical findings –

APPENDIX II

Staining procedure:

Periodic Acid Schiff stain:

- Bring section to water
- Rinse in distilled water
- Treat with Periodic Acid solution for 5 min
- Rinse in distilled water
- Place in Schiff reagent for 15 min
- Place in running tap water for 10 minutes for pink colour to develop
- Stain in Harris haematoxylin for 6 minutes
- Rinse in tap water
- Differentiate in acid alcohol for 3-10 quick dips
- Wash In tap water
- Dip in lithium carbonate
- Wash in tap water
- Dip in alcohol 95%
- Absolute alcohol 2 changes
- Xylene 2 changes
- Mount in DPX.

Result

- PAS positive substance-Magenta colour
- Nuclei-Blue

RETICULIN STAIN:

- Bring sections to water
- Potassium permanganate 1% for 2 minutes.
- Wash in tap water
- 1% oxalic acid for 3 minutes
- Wash in water
- Silver nitrate solution for 10 minutes
- Rinse in distilled water for 10 seconds and in 10% formalin for 3 minutes
- Tone in goldchloride 0.1% for 10 minutes
- 1% Oxalic acid for 1 minute
- 1% sodium thiosulphate for 1 minute
- Wash in water
- Dehydrate in absolute alcohol
- Clear in xylene
- Mount in DPX

Result :

- Reticulin fibres-Black in colour.

KEY TO MASTER CHART

M - MULTIPARITY

N - NULLIPARITY

UL - UNILATERAL

BL - BILATERAL

ABD.MASS - ABDOMINAL MASS

ABD.PAIN - ABDOMINAL PAIN

SEROUS CYSTADENOCARCINOMA PD- SEROUS CYSTADENOCARCINOMA-POORLY DIFFERENTIATED TYPE

SEROUS TUMOUR-BL - BORDERLINE SEROUS TUMOUR

CK - CYTOKERATIN

EMA - EPITHELIAL MEMBRANE ANTIGEN

AFP - ALPHA FETO PROTEIN

PAS	-	PERIODIC ACID SCHIFF
WT-1	-	WILMS TUMOUR ANTIGEN